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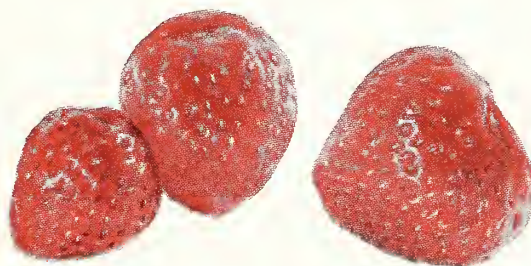
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No. 1

## Reduplication Of The Stomach A Case Report

EUGENE E. O'DONNELL, M.D. and GERALD LEARY, M.D.\*

M.C., a 26 year old white married female, was admitted to the Mercy Hospital on 1/21/63.

Since approximately 13 years of age, she had had epigastric distress after meals without nausea or vomiting, which was usually relieved by belching gas. At that time, x-ray studies of her upper gastrointestinal tract were interpreted as negative. An upper G.I. series was repeated at the age of 18 years because of persistence of these symptoms, and these were likewise negative.

For about two years before admission, post-prandial distress had increased in severity, again without nausea or vomiting or hyperacidity. On 8/14/61, upper gastrointestinal x-rays were again carried out with negative results. On 12/17/62, x-ray examination of the stomach disclosed a filling defect in the antrum of the stomach, which was constant when repeated two days later. The Graham-Cole test was normal. Past history was otherwise negative except for an uncomplicated appendectomy in 1954.

Upon admission, the physical examination was that of a well-developed and well-nourished young adult female, not acutely ill. There was mild discomfort, on deep palpation, in the epigastrium. Laboratory studies disclosed a mild secondary anemia. Stool examinations were negative for occult blood.

On 1/24/63, laparotomy was performed through a midline incision extending from the xyphoid to the umbilicus, with a preoperative diagnosis of a gastric polyp. A firm nodule in the prepyloric portion of the anterior stomach wall was palpable as a mass about 1



FIG. 1. Filling defect of gastric antrum.

cm. in diameter. The gastro-hepatic and gastro-colic ligaments in this area were divided between clamps and the bleeding points ligated. The antrum of the stomach was resected, and a Billroth I anastomosis performed, using #00 chromic intestinal suture for the mucosa and

\*From the Mercy Hospital, Portland, Maine.





FIG. 2. Cyst lined by gastric mucosa.



FIG. 3. Normal gastric mucosa lining the cyst.



FIG. 4. Superficial ulceration of gastric mucosa overlying the cyst.

interrupted silk sutures for the serosa. The abdominal incision was closed anatomically.

The postoperative convalescence was uneventful, and the patient was discharged from the hospital on the 10th postoperative day. A follow-up visit since that time shows that she has been entirely relieved from the gastrointestinal symptoms.

On pathologic examination a slight elevation of the mucosa of the stomach in the prepyloric area was encountered in the center of which a small ulceration was noted. Sections through this elevated area through the entire stomach wall revealed a 1 cm. cyst in the gastric wall. The cyst contained mucus. No connection between the lumen of the cyst and the gastric lumen could be demonstrated.

Microscopic examination disclosed a cyst lined by normal gastric mucosa surrounded by smooth muscle. Beyond the smooth muscle were some ducts lined by mucus secreting columnar epithelial cells. The depression noted on the overlying gastric mucosa was a small superficial erosion. This case is reported because it is an unusual anomaly.

In 1962 Anderson, Silberman and Shields collected 63 cases of alimentary duplication recognized after the age of 13 years. These cases were compiled from the world literature from 1949-1959. Twenty-six cases originated from the esophagus, 9 from the stomach, 6 from the duodenum, 10 from the small intestine, 5 from

the colon, 3 from the esophagus and stomach and 2 from the small and large intestine.

The symptoms of gastric duplication were usually vague, suggestive of peptic ulcer disease. Nausea, pain and occasional vomiting occurred. Gastrointestinal bleeding was less common.

Gross reported on 68 duplications of the alimentary tract at the Boston Children's Hospital. Two of these were in the stomach. He stated that duplications of the stomach usually do not cause obstruction. The complaints have been those of epigastric fulness. An abdominal mass may be palpable.

Stout suggested that almost all gastric duplications probably are due to developmental defects.

McLetchie felt that vertebral anomalies such as spina bifida or vertebral clefts associated with some cases of alimentary duplications might be responsible for the defect. In our case no spinal abnormalities were demonstrable.

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# Maple Syrup Urine Disease\*

SELMA E. SNYDERMAN, M.D. and L. EMMETT HOLT, JR., M.D.

This condition was first described by Menkes, Hurst and Craig in 1954; they reported 4 siblings who died during the first six weeks of life with a progressive neurological disorder, whose urine exhibited a characteristic odor resembling that of maple syrup. The disease was identified as a disorder of the metabolism of the branched chain amino acids by Westall, Dancis and Miller in 1957, the exact location of the metabolic block being subsequently located by observations made in 3 different laboratories. More than 25 cases of this disorder are now known. It appears to be transmitted as an autosomal recessive. The disease has been recognized in several parts of the United States, England, Germany, Greece and Japan. With one exception, untreated patients have all succumbed in early infancy; one patient is known to be alive at the age of 8 years, grossly retarded and with severe neurological symptoms. One other patient exhibited retrogression of the symptoms and biochemical abnormalities in late infancy; it is a question whether this case represents a variant of the disease.

The symptoms usually start soon after birth; they may be evident before the end of the first week; there is anorexia, failure to thrive, hypertonicity and at times convulsions and a semicomatose state. With the development of symptoms the characteristic odor of the urine becomes apparent; it has been described as "malty," "like caramelized sugar" or like maple syrup. "Oast house disease" (Smith and Strang) is presumably the same entity, although their patient also suffered from phenylketonuria. At times other secretions exhibit this odor; it has been described in perspiration and in the cerumen of the ear.

The basic metabolic defect involves the branched chain amino acids — leucine, isoleucine and valine (Fig. 1). These are transaminated in a normal manner with the formation of the corresponding keto acids; the keto acids, however, are not further degraded by oxidative decarboxylation and they as well as the amino acids themselves accumulate in the blood and are excreted in excess in the urine. An excess of branched chain acids has also been demonstrated in the spinal fluid. The enzyme defect has been demonstrated by Dancis in the white blood cells and the skin fibroblasts. Hydroxy derivatives of the branched chain amino acids have been identified in the urine. A secondary alteration of tryptophan metabolism similar to that observed in phenyl-

ketonuria, Hartnup disease and several other conditions has also been observed in maple sprup urine disease; this consists in increased production of tryptophan derivatives formed by transamination, notably indolacetic acid and indolactic acid. When a corrective diet low in branched chain amino acids is instituted the abnormality of tryptophan metabolism disappears.

The finding of alloisoleucine in the plasma and urine of these children is of some interest. In the original analysis of plasma by column chromatography an abnormally large peak was observed in the region of methionine and an abnormality of methionine metabolism was postulated. However, improved techniques employing a more sensitive resin revealed 2 peaks in this region, a normal methionine peak and a large unknown peak which was subsequently shown to be alloisoleucine. The alloisoleucine is formed from isoleucine, apparently by enolization of the keto derivative g-keto, B methyl valeric acid and subsequent reamination which results in the formation of both isoleucine and alloisoleucine (Fig. 2). The administration of isoleucine causes a prompt rise in the plasma isoleucine followed by a delayed rise in alloisoleucine; the latter persists in the blood for a long time because of a poor renal clearance.

The metabolite responsible for the peculiar odor has not been definitely identified; it appears to be related particularly to isoleucine, since it is brought out by a load of isoleucine, but not by a load of the other two branched chain acids. The exact cause of the neurological damage is likewise obscure. A direct toxic effect of the branched chain acids themselves or of their derivatives is one possibility. Toxic neurological symptoms develop readily after a lapse of dietary control and also after a load test with leucine. Tashian has shown in rat brain homogenates that the activity of the enzyme glutamic acid decarboxylase is markedly inhibited by the keto derivatives of leucine and valine. A second possibility is that the elevated levels of the branched chain amino acids interfere competitively with the uptake of other amino acids by the brain cell. It has also been suggested that the secondary abnormality of tryptophan metabolism is the cause of the brain damage with increased transamination and decreased 5-hydroxylation may be responsible for reduced serotonin levels in the brain.

It is difficult to relate the pathological changes to the biochemical abnormalities. There is a defect of myelin formation throughout the brain. This is accompanied by areas of spongy change, astrocytosis and a decrease of oligodendrocytes. Pathological changes in the neurones have not been demonstrated.

\*This paper was presented by Dr. L. Emmett Holt, Jr., professor of Pediatrics in New York University, at the Medical Conference at the Pine'land Hospital, Pownal, Maine on July 11, 1963.

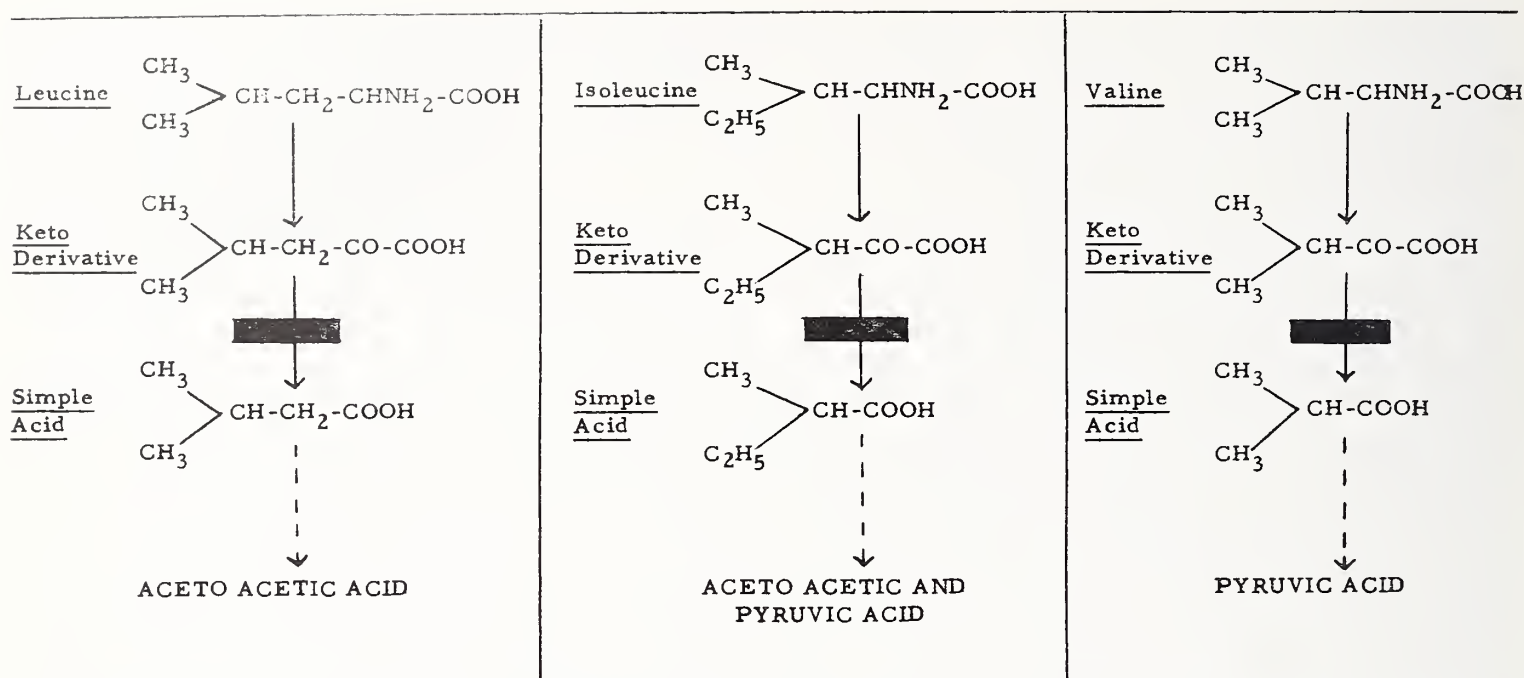


FIG. 1. Catabolism of Branched Chain Amino Acids Illustrating the Metabolic Block in Maple Syrup Urine Disease

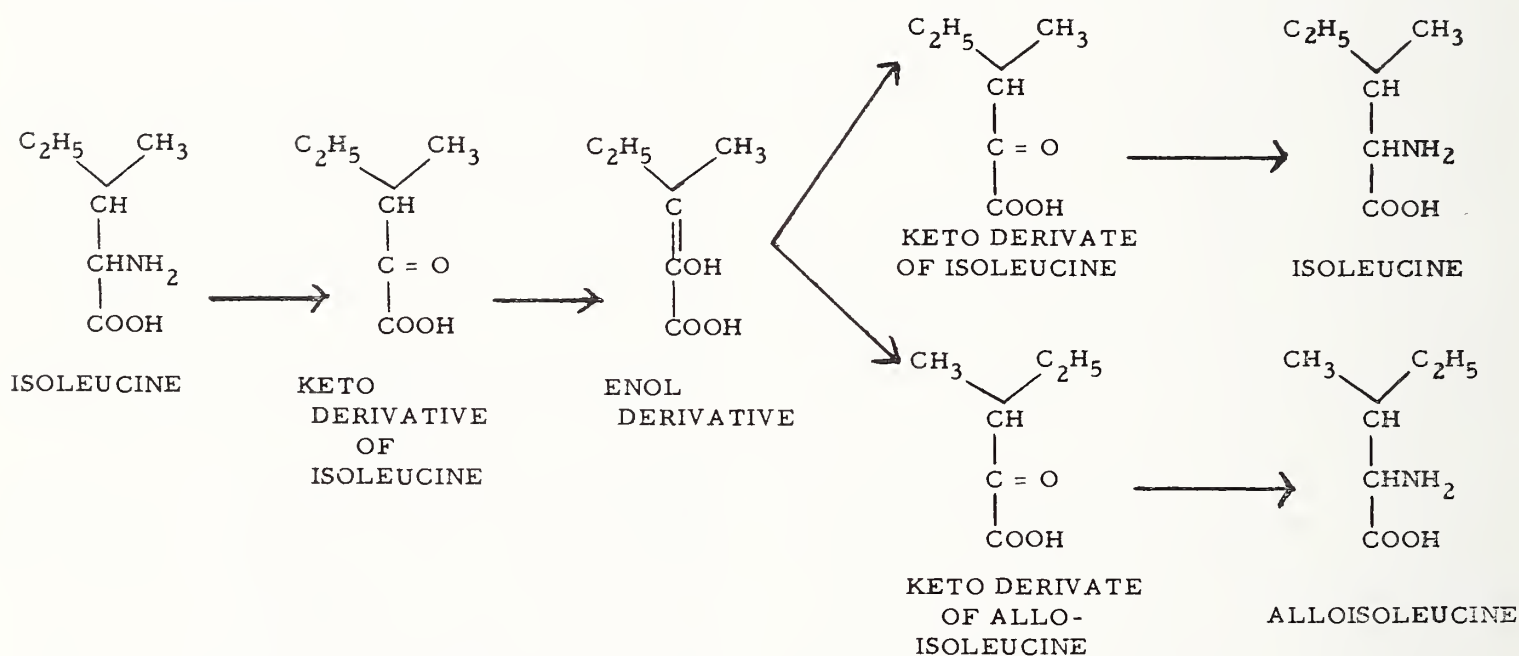


FIG. 2. Showing Formation of Alloisoleucine From Isoleucine in Maple Syrup Urine Disease

Dietary restriction of the three branched chain amino acids will correct the biochemical abnormalities and has resulted in striking clinical benefit. When given sufficiently early in the disease there are indications that the damage to the nervous system may be prevented, the benefit being less striking the longer therapy is delayed. The writers have had under treatment one such patient who developed symptoms at 6 days of age and whose diet was corrected at the age of 14 days. This patient, now 4½ years of age, is in good physical health and shows no neurological symptoms; she is, however, somewhat retarded mentally, and has had a poor growth performance. It is possible that the retardation is related to nutritional factors because of prolonged restriction of

methionine undertaken to reduce what had been regarded as a high methionine level. During the past year her growth and intellectual development have been accelerated. A second child treated by Dent and Westall from the age of 5 days, and not deprived of methionine, is said to be developing normally at the age of 18 months. The importance of early diagnosis in these patients cannot be overemphasized. It may well be that if one waits for the urine to show the characteristic abnormality — a positive reaction for keto acids with dinitrophenyl hydrazine — some damage to the nervous system may already have occurred. An earlier diagnosis can be made by following the plasma levels of the branched chain acids by column chromatography or by identifying the



enzyme deficiency in the white blood cells. Such measurements are not likely to be carried out except in the case of siblings of affected patients.

The problem of dietary control is not a simple one since natural foods are all well supplied with branched chain amino acids. It is therefore necessary to supply the major portion of the diet as a synthetic mixture, the nitrogen of which is furnished as a mixture of pure amino acids in which the branched chain acids are kept low, their intakes being adjusted at intervals to maintain normal plasma levels. This necessitates frequent analyses of plasma during the early months of life. We have found that it is possible to give these patients a small amount of milk, which will furnish the bare minimum quantities of the branched acids which are needed for growth; the bulk of the diet must, however, be synthetic. In the initial period, however, when the diagnosis is first made and the branched chain acids are markedly ele-

vated, it is desirable to reduce their concentration promptly by using a completely synthetic diet for a week or two.

A difficulty in the management of these patients has been that of intercurrent infections. At such times food intake may be curtailed and the patient be forced to live on his own tissues which contain an abundance of branched chain acids; as a result their levels rise promptly in the blood and there may be recrudescence of symptoms and of the maple syrup odor. It is therefore of the greatest importance to protect these children from infection and to maintain their food intake during such infections.

With early detection and facilities for dietary regulation it appears possible to protect the nervous system in these patients. The recognition of heterozygote carriers of the trait, if that proves possible, will go far to make early diagnosis possible.

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# HELPLESS . . . But Not Hopeless

RICHARD H. WHITTEMORE\*

Some alcoholics are sicker than others.

Some are not ready for treatment yet.

Professional and nonprofessional Alcoholic Rehabilitation workers (teams) and others from time to time wonder why some alcoholics recover rapidly, some slowly and painfully, and why others get progressively sicker. Yet all are exposed to the same techniques and personnel. At times one is tempted to say "many are *hopeless*." But when we examine our records we can find many *recovered* or *recovering* alcoholics whom we had mentally classified as "hopeless." Shouldn't we regard all alcoholics as temporarily *helpless* rather than *hopeless*?

I would like to describe this challenging group of problem drinkers who do not *seem* to respond to our alcoholic rehabilitation treatment techniques. This group can be broken down into three categories, all of which include men and women of various ages, faiths, ethnic backgrounds, educational opportunities and "social levels":

1) Those persons who have deep and serious mental and emotional illnesses for which their excessive drinking patterns are but a symptom. (It is questionable as to how many in this category can be classified as "full fledged alcoholics.")

2) Those persons who are *extremely* sensitive and immature in a world filled with "cruel people, tough problems and unpleasant obligations." In this category we find a group of *typical* but *sicker* alcoholics.

3) Those persons who are not sick enough; who have not suffered enough; who are not ready to accept treatment. They firmly believe they have control over their use of alcohol. They don't want to give up drinking in spite of the problems that are starting to plague them.

In all three categories we recognize one common denominator: All are using alcohol to excess, in various forms, *as a drug* . . . not as a beverage? They drink to escape pain – inner – outer – and unidentified. Their motives for coming to an alcoholic rehabilitation center all differ.

In the *first category* we find many sick people who benefit little from outpatient counseling and group therapy. When these patients do not respond, and deeper mental and emotional problems are recognized, we refer them to psychiatric treatment or to a mental hospital or institution for longer term inpatient treatment. If he refuses, the family is made aware of its responsibility in the matter. Those who do complete treatment in mental hospitals are encouraged to continue with outpatient

therapy at our centers. However, there are always those few incurable psychopaths who never learn from experience, and there are those too with permanent brain damage. But as stated in chapter five of the book *Alcoholics Anonymous*: "Their chances are less than average" . . . "but many of them do recover if they have the capacity to be honest." Even though everything humanly possible fails in treating a problem drinker, whether his pattern be excessive, addictive, steady or periodic, regardless of the seriousness of cause and diagnosis, we must never rule out the possibility of recovery through "a spiritual awakening" . . . rare, but very real. Experience, understanding, patience and hope are our most valuable tools in working with this *helpless* type of problem drinker.

In the *second category* we have "unruly children" running loose without guardians. They eat too many green apples and too much candy because they are undisciplined. In spite of the "tummy aches" they behave this way because they enjoy stealing, lying, overindulging, breaking rules, and boasting about all of this. They are rebels concerned only with their own selfish pleasures and desires.

But usually we are dealing with men and women over twenty-one; human beings unprepared for adult responsibilities, incapable of facing life's problems, and unwilling to conform to society's image of adult behavior. This brings on reprisal, embarrassment, dissolution, and antisocial behavior . . . Drinking.

These extremely immature men and women innocently begin to use alcohol as a "magic potion" either to help them grow up and act like adults, or to escape from reality entirely into their own world of fantasy. Only those with the metabolic tolerance for, and the emotional satisfaction from alcohol, succeed in becoming alcoholics.

After years of excessive use these people become addicted to, and completely dependent upon the use of alcohol in various forms. When they appear for treatment they have very little courage, incentive or strength for the uphill road to recovery. If a few weeks of sobriety *isn't* paying immediate dividends according to their expectations, they become easily discouraged, and with a "good alibi," run for the bottle.

A great many included in this second category of *tough cases* have no real purpose in life. They have not found a reason for existing. They feel they have little to offer, no service to perform . . . no causes to champion. After years of drinking they reach a stage of "suspended

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vegetation" on a cloud of liquid spirits. Without motive there is no purpose; without purpose there is no action; without action there is no recovery.

Alcoholics Anonymous has had some success with this type by providing a purpose and a society to fit their needs. In A.A. groups they can find *acceptance* and a chance to be of *service* to others they can understand, but many of them will not affiliate with A.A.

One could go on and on in detail about those in this second category because they are the most frustrating to a counselor, and at the same time . . . the most challenging. This group is *helpless*, but far from *hopeless*. Almost overnight their attitudes can change. Because, like little children they are impressed by example, enthused as easily as they are disappointed. During every period of sobriety they grow up just a little more.

Time, patience and sobriety can level them off. On the other hand, such things as a love affair or sudden wealth can cause a relapse. No matter how often they slip they must be encouraged to try again. They need constant reassurance and guidance, and the association of recovered alcoholics. When in distress, they must feel free to come to the counselor for private consultation.

Naturally, we look for and find the guilt and inferiority complexes in group two. And there is always the risk in both groups one and two of a few becoming *institutionalized* if they are type who adjust well to hospital or jail routines. Shut off from temptation in a world that provides for their basic needs, they find protection, acceptance and relaxation. They make friends easily and receive special treatment from the keepers or attendants. When they view confinement not as punishment, but as a gift from society, they will accept it in preference to "the struggle." Here they cannot fail.

These first two groups also produce those with sex deviations, frustrations and obsessions. In the first group we find the nymphomaniacs and homosexuals. The second group becomes more complicated as their problems are well hidden. But we detect in some . . . latent homosexuality, masturbation guilt, drives for sexual security on any level, obsessions to prove virility, the pride in denomination and conquest, the desire for the thrill of new sex experiences which serve as a temporary euphoric, and the seeking of acceptance and affection in sexual relations. Then there are the timid ones who find only rejection and frustration.

Those in group two seldom use reason. They can only live with themselves by rationalizing. For them alcohol releases inhibitions, provides excuses for anti-social behavior, and drugs the conscience. Sobriety is not easily gained or accepted. They are very sick people.

We must not allow those in *category three* to upset the programs and techniques we have used successfully with the other types of problem drinkers and alcoholics. This group is just not ready to give up the bottle or to accept treatment for a habit. They are a selfish, care-free, deceitful, troublesome lot; inflated with ego and

self-importance; stubborn, headstrong, and determined to have their own way. *They believe they are not alcoholics and they resent being referred to as such.*

These men and women enjoy everything about drinking. Without a drink in their hands they have little face, character or identity. When drinking they are ten feet tall. They like the comradeship, the bottles, the glasses, the ice, the mixers, the noise and confusion. They don't enjoy drinking alone. They must have an audience.

Perhaps these indictments seem a little strong, but we are dealing here with people who are strong in their desires and convictions. They suffer from a warped sense of values, have great limitations, and misconceived opinions of society in general. Many are brilliant professional men and women. Some have no financial worries, and those who do, won't admit it because the ego says they will soon be on top. These problem drinkers do not believe they are alcoholics "because I can stop drinking whenever I want to. I just don't want to." They cannot identify themselves with other alcoholics, drunk or sober.

Persons in this group enter our rehabilitation centers only under pressure from home, the employer, or the courts. Very few listen, or are willing to learn or try. Most of them spend their time trying to convince us that they are not alcoholics, that they have been misunderstood and abused; that they are too busy and too important to even be in a rehabilitation center; that they admire our work and can see the good in it for others. Need I go further in describing this type?

We must admit that we have very few scientific facts about the ramifications of alcoholism and the alcoholic personality. But it seems to be generally accepted that *alcoholism is a progressive disease*. Thus, if we practice tolerance and patience, show a little sympathy and understanding; sow a few seeds of information, and make a few constructive recommendations, we may get the chance in later years to really help these people when they are ready to surrender.

In conclusion, I would say that roughly half of those men and women who enter our alcoholic rehabilitation centers, clinics, or counseling offices, fall within the three categories herein described in brief, general terms. The "Alcoholologist" still searches for words to describe causes, symptoms, types and techniques in this relatively new field of social and/or public health work. The other fifty percent of excessive drinkers not discussed in this article, are sick too, but they are much more receptive and cooperative, and are sincerely willing to try to overcome their problems with guidance. Through counseling, group therapy, the fellowship of Alcoholics Anonymous, or a combination thereof, their cases are much improved, or arrested altogether.

I have seen enough so called "hopeless alcoholics" recover by one means or another to indicate to me that in our work there should never be a file marked: "HOPELESS."



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## Causes Of Disability — Aid To Disabled Program

EDSON K. LABRACK, M.P.H.\*

In October, 1962, the Department conducted a special study of certain personal and financial characteristics of recipients of assistance under the Aid to Disabled Program in Maine. Such a study was required of all states by the U. S. Department of Health, Education and Welfare.

At the time of the study there were 2,264 persons in Maine who received assistance under the program. Data for the study was obtained from a systematic sample of 576 cases, or 25.0% of the total caseload. Data in this report are estimates for the total caseload based on the sample.

### GENERAL CHARACTERISTICS OF RECIPIENTS

About 60% of the recipients were males. There were 1,289 male recipients and 975 female recipients. Only 47, or 2.0% of the recipients were nonwhite. Less than 1.0% of the population of the State is nonwhite according to the 1960 Census of Population. Recipients ranged in age from 18 to 64 years. Upon reaching age 65, disabled recipients are automatically transferred to the old-age assistance program. The median age for recipients was 53.5 years.

### IMPAIRMENTS CAUSING DISABILITY

In order to qualify for assistance under the aid to disabled program the applicant must be a needy person who has a condition which reduces his ability to function as a wage earner or homemaker and which condition is total and permanent. The applicant must be examined by a physician whose findings are reviewed by the Medical Review Team of the Division of Family Services.

Nearly one half (1,101) of the recipients had two or more disabling conditions listed. In such cases the more severe condition is listed as the "primary impairment" and a second condition as the "secondary impairment." Only two conditions were selected for tabulation. Undoubtedly the selection of primary and secondary conditions tends to be arbitrary in certain cases where two or more approximately equal problems exist.

Five leading causes were responsible for disability in nearly one half (49.2%) of all cases, and 10 leading

causes were responsible in over two thirds (68.0%) of all disabled cases.

Mental retardation was the most important primary impairment. Mental retardation was the primary impairment in 522, or 23.0% of all cases. In addition there were 106 other cases where mental retardation was listed as a secondary impairment, so that mental retardation was a significant factor in the disability of at least 27.7% of the caseload. Undoubtedly it was a factor in other cases which had other severe disabling conditions listed as primary and secondary.

Arthritis ranked second among primary impairments. There were 211 cases (9.3%) whose primary impairment was arthritis. In all, there were 290 arthritics receiving assistance including those with arthritis as a secondary impairment. This was 12.8% of the total caseload.

The third most important cause of disability was chronic degenerative heart disease (arteriosclerotic heart disease, coronary disease, chronic valvular heart disease, and chronic myocarditis). One of these conditions was the primary impairment in 156, or 6.9% of all cases.

Vascular lesions of the central nervous system and accidental injuries ranked fourth and fifth, being responsible for the disability of 118, or 5.2% and 110, or 4.8% of all recipients respectively. Other leading primary impairments were: (6) Hypertensive heart disease, 102, 4.5%; (7) Cerebral spastic infantile paralysis, 98, 4.3%; (8) Pulmonary emphysema, 90, 3.9%; (9) Tuberculosis, 79, 3.5%; and (10) Congenital malformations, 59, 2.6%.

Table I shows the distribution of selected primary and secondary impairments among recipients.

There were 1,175 recipients with impairments which may be classified in more specific terms than the disease or injury responsible; e.g.: deafness, missing limb, etc. The most common of these impairments was a defect or deformity of the limbs, spine, or trunk. There were 499 recipients with these defects, or 22.0% of the entire caseload. Paralysis, including cerebral palsy, was present in 271, or 12.0% of all recipients. An additional 181 (8.0%) had serious speech defects, 79 (3.5%) were blind or near blind, 59 (2.6%) had impaired hearing, 39 (1.7%) had a missing arm, hand, or leg, and 31 (1.4%) had missing fingers or toes.

\*Director of Research and Vital Records.



Table 1: Recipients of APTD (Aid to the Totally and Permanently Disabled) by primary and secondary impairments causing disability: Maine, October 1962.

Disease or condition	Number of recipients	
	Primary impairment	Secondary impairment
Total	2,264	1,101
Tuberculosis, all forms (001-019)	79	16
Syphilis and its sequelae (020-029)	24	8
Late effects of poliomyelitis (081)	35	—
Malignant neoplasms (140-205)	35	4
Asthma (241)	24	16
Diabetes (260)	51	35
Mental deficiency (325)	522	106
Other mental, psychoneurotic, and personality disorders (300-324, 326)	20	51
Multiple sclerosis (345)	12	—
Paralysis agitans (350)	28	4
Cerebral spastic infantile paralysis (351)	98	16
Vascular lesions of CNS (330-334)	118	74
Epilepsy (353)	31	62
Other diseases of central nervous system	83	16
Rheumatic fever and chronic rheumatic heart disease (400-416)	28	16
Chronic degenerative heart diseases (420-422)	156	58
Other diseases of heart (430-434)	47	28
Hypertensive heart disease (440-443)	102	8
All other diseases of circulatory system (444-468)	44	90
Pneumoconiosis and bronchiectasis (523, 524, 526)	20	4
Pulmonary Emphysema (527)	90	43
Other diseases of respiratory system	16	47
Diseases of digestive system (530-587)	31	59
Arthritis (720-725)	211	79
Deformities (745-749)	47	24
Osteomyelitis and other diseases of musculoskeletal system (730-744)	63	50
Congenital malformations (750-759)	59	20
All other diseases	60	124
Accidental injuries (N800-N999)	110	43

ORIGIN OF IMPAIRMENT

The origin of the primary impairment was attributed to disease processes in 1,356, or 60.0% of the cases. Congenital malformation and birth injury ranked second in importance with the primary impairment being attributed to these causes in 727, or 32.1% of all cases. The primary impairment was attributed to injuries in 95, or 4.2% of the cases. Twenty of the latter (0.8%) were employment injuries. In 86 cases the origin of the impairment was unknown or not stated.

Data concerning duration of impairments was available in 1,100 or slightly under half of the cases. Despite deficiencies in available data it is apparent that the disability of most recipients tends to be of long duration. In 153, or 6.8% of all recipients, the primary impairment had been present for less than 10 years. In 841, or 37.1% of the cases the primary impairment had been present for 20 years or more.

MOBILITY AND CARE FROM OTHERS

Disability ranged among recipients from those who are completely bedfast to those who are able to get about outside the home by themselves. In 617, or 27.2% of the cases the recipient was confined to the home. In 538 of the cases where the recipient was confined to the home, or 23.7% of all cases, the recipient needed care from others. Among the latter were 94 (4.2%) who were bedfast and 177 (7.8%) who were chairfast. An additional 244 (10.8%) were not confined to the home because of their physical or mental conditions, but needed care in the home. There were 1,214 (53.6%) recipients who were able to get about in or out of the home without assistance.

SUMMARY

The APTD program had 2,264 recipients in October 1962. Mental retardation was the chief cause of disability followed by arthritis and degenerative heart diseases. Most recipients had been disabled for a long period of time. About one half of the recipients were able to get about by themselves without help.

Annual Meeting Dates For Your 1964 Calendar

Maine Medical Association, June 14-16, 1964 at The Samoset, Rockland, Maine.

American Medical Association, June 21-25, 1964 at the Fairmont and Mark Hopkins hotels and Civic Auditorium, San Francisco, California.

## Report Of Maine Medical Association Delegate On Actions Of The House Of Delegates Of The American Medical Association At The Seventeenth Clinical Meeting, Portland, Oregon, December 1-4, 1963

The house at its opening session expressed deep shock at the tragic death of the late President John F. Kennedy and directed that a letter of heartfelt sympathy be sent to Mrs. Kennedy, her children and the late President's family. The House also pledged its support to President Lyndon B. Johnson in forging national unity in the weeks and months ahead and offered the Association's resources, counsel and cooperation on matters of health.

Dr. Edward R. Annis, AMA president, reporting on the recent House Ways and Means Committee hearings on the King-Anderson Bill, told the House:

"The combined testimony of the American Medical Association, the state societies and our allies made a far greater impact on the members of the committee, friend and foe alike, than at any other time in the history of this long and bitter conflict."

Dr. Annis also reported that under questioning from Committee Chairman Wilbur Mills, actuaries of the Department of Health, Education and Welfare admitted that the program of tax-paid hospitalization and related benefits for the aged proposed in the King-Anderson Bill would require a tax rate twice as high as they have previously claimed.

The House approved a Board of Trustees proposal that the American Medical Association Education and Research Foundation undertake a "comprehensive program of research on tobacco and health."

Agreeing that many gaps exist in knowledge about the relationship between smoking and health, the House declared that the study should be "devoted primarily to determining which significant human ailments may be caused or aggravated by smoking, how they may be caused, the particular element or elements in smoke that may be the causal or aggravating agent, and methods for the elimination of such agent."

The action called for procuring a project director "whose experience, qualifications and integrity will assure that such a research project will be conducted effectively, exhaustively and with complete objectivity."

The House agreed that the project should be financed by a substantial contribution from the American Medical Association and that contributions should be solicited from other sources — industry, foundations, voluntary health agencies and physicians. It was emphasized that contributions will be accepted only if they are given without restrictions.

Subsequent to the House action, the AMA Board of Trustees voted to contribute \$500,000 to help finance the research program.

The House considered two proposals related to Negro physicians — a Board report on hospital staff privileges and a resolution concerning membership eligibility in state and county medical societies. The board report was approved, but the resolution was not adopted.

In adopting the Board report, the House declared that "members of the medical staff of every hospital, where the admission of physicians to hospital staff privileges is subject to restrictive policies and practices based on race, be urged to study this question in the light of prevailing conditions with a view to taking such steps as they may elect to the end that all men and women professionally and ethically qualified shall be eligible for admission to hospital staff privileges on an equal basis, regardless of race."

In both its approval of the Board report and its rejection of the proposed resolution — which would have denied the rights and privileges of AMA membership to members of any state or county society which refuses membership to any qualified physician because of race, religion or place of national origin — the House reaffirmed 1950 and 1952 policy actions on this subject and directed that a copy of the 1950 resolution again be sent to each state and county medical society. That resolution urged that "constituent and component societies having restrictive membership provisions based on race study this question in the light of prevailing conditions with a view to taking such steps as they may elect to eliminate such restrictive provisions."

In approving a Board report on professional relationships with voluntary health agencies, the House declared that the "AMA maintain its policy of neither approving nor disapproving national voluntary health agencies." It also agreed "that the AMA, through its Committee on Voluntary Health Agencies, maintain its position of offering guidance on medical aspects of national voluntary health agency programs."

The House adopted a policy statement pointing out that in recent years there has been a dramatic growth of blood banking facilities in the United States and declaring that, "It is highly essential that the organization of new blood banking programs and the modification of existing ones should have, in the interest of public health and safety, the approval of the county or district medical society and, therefore, should be coordinated with existing approved blood banking facilities." The House also approved a floor amendment stating that since a blood bank can well be considered a medical facility, the top authority in a blood bank should be a physician.

Extended AMA Affiliate Membership to *scientists* in sciences allied to medicine;

Approved an amendment to the Bylaws which would permit the *opening session* of the House of Delegates to be held on Sunday afternoon or evening;

Expressed gratification that the work of the Committee on *Medicine and Religion* has received widespread acceptance and support from state and county

*Continued on Page 16*



in virtually all diarrheas...prompt symptomatic control

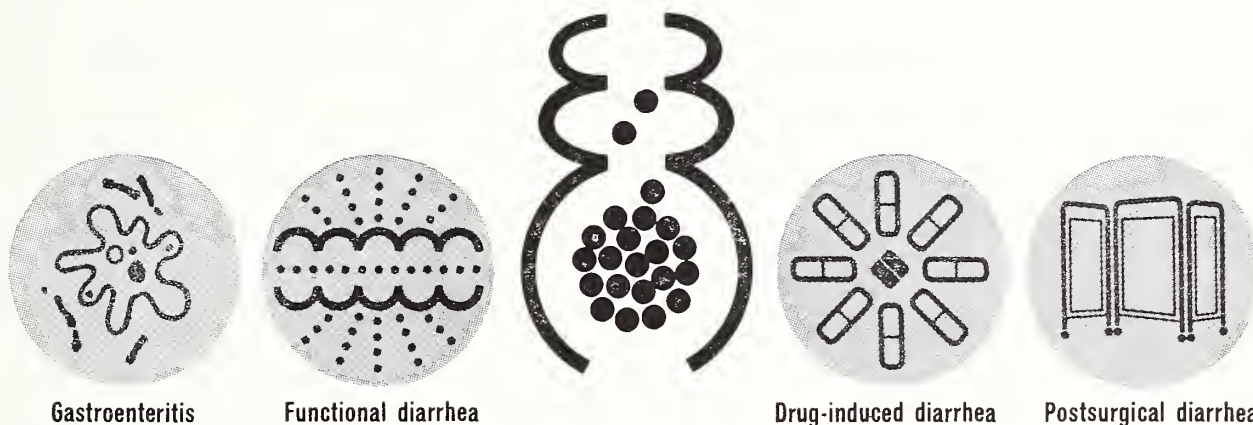
# LOMOTIL<sup>®</sup>

TABLETS / LIQUID—Each tablet and each 5 cc. of liquid contains:

diphenoxylate hydrochloride . . . 2.5 mg.

(Warning: May be habit forming)

atropine sulfate . . . . . 0.025 mg.



Gastroenteritis

Functional diarrhea

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Lomotil controls the basic physiologic dysfunction in diarrhea—excessive propulsive motility. Pharmacologic evidence indicates that it does so by directly inhibiting propulsive movements of the intestines. This direct, well-localized activity controls diarrheas of widely varied origin and does so promptly, conveniently and economically.

The relatively few conditions in which Lomotil has given less than satisfactory control have been, for the most part, those such as severe ulcerative colitis in which too little anatomic or functional capacity of the intestines remains for the motility-lowering action of Lomotil to have effect.

It should be noted, however, that Lomotil has proved highly useful in mild to moderate ulcerative colitis and in several other refractory forms of diarrhea.

*The recommended initial adult dosage is two tablets (2.5 mg. each) three or four times daily, reduced to meet the requirements of each patient as soon as the diarrhea is controlled. Maintenance dosage may be as low as two tablets daily. Children's daily dosage (in divided doses) varies from 3 mg. for a child of 3 to 6 months to 10 mg. for one 8 to 12 years of age. Lomotil is an exempt narcotic; its abuse liability is low and comparable to that of codeine. Recommended dosages should not be exceeded. Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdose.*

*Research in the Service of Medicine*

SEARLE

## *Maine Heart Association Notes*



### **Rehabilitation Of The Cardiac Patient**

“. . . We as physicians must view heart disease in all of its ramifications. Heart disease is primarily a chronic process. . . .

“Care is required for the patient over months and years. . . .

“. . . it is not enough merely to stop the advance of disease. One must endeavor to restore the patient to the full life. This is rehabilitation. This philosophy should be the credo of all physicians and of all the ancillary professionals dealing with a cardiac patient incapacitated by his disease. Man is a complex creature with a mind and emotions, and illness affects him in his totality – mind and body besides heart and blood vessels.

“Rehabilitation to be effective must be complete. It must attempt to attack all aspects of the situation. . . . It involves an expert analysis of the patient’s mental attitude to his disease, the unraveling of the hidden fears that reside in him and his family. It requires the instillation of hope that he will attain within the limits of his body . . . his birthright to happiness of the full life and gainful occupation in his job that is interesting and challenging.

“We are just at the beginnings of the science of rehabilitation. The day must soon come when no alert community can neglect this important area, when no medical center is considered to be really a center unless it has an adequate rehabilitation program, and when this program reaches into every home, rich and poor alike. It will then be accepted that in treatment we do not stop by merely making people well, but well and useful.

“We must stress as the aim of good medicine, the restoration of human beings to their normal role in family, in their society, and at their work. This is no investment in bricks and mortar; it is an investment in human beings.”

Reference: Katz, L. N., et al. *Circulation*, 17, pages 114-126, 1958.



# County Society Notes

## ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held at St. Mary's General Hospital in Lewiston, Maine on October 17, 1963. The meeting was called to order by the President, Morris E. Goldman, M.D. with twenty-nine members present.

The film, *Operation Hometown*, narrated by Edward R. Annis, M.D., President of the American Medical Association was shown, following which John W. Carrier, M.D., Chairman of *Operation Hometown*, introduced Mr. Maurice Kramer, Regional Representative for the New England States, who has recently joined the AMA Staff.

Merrill S. F. Greene, M.D. spoke of the need for a fund for needy members and other emergencies. It was voted favorably that the President appoint a committee to study the advisability of having a "Welfare Fund"; and if considered feasible, the ways and means to implement such a fund.

DONALD L. ANDERSON, M.D.  
*Secretary*

## CUMBERLAND

A meeting of the Cumberland County Medical Society was held at the new Nursing Pavilion and Jewish Home for the Aged in Portland, Maine on October 17, 1963. After a social hour and dinner, the meeting was called to order by the President, Philip P. Thompson, Jr., M.D.

Mr. Saul Chason, President of the Home, greeted the group; and Mr. Jules Krems, Executive Director, gave a brief resumé of the aims and policies of the new nursing pavilion in regard to services and admissions in particular. He emphasized that the Home was to be non-profit and non-sectarian, and an appreciable proportion of the costs would be borne by legacies and voluntary contributions.

The obituaries of Drs. Albert D. Foster and Hans V. Mautner were read and it was voted that these be spread on the minutes of the society and copies be sent to the families.

The program of the evening was *Operation Hometown*. Howard P. Sawyer, Jr., M.D. gave a very informative and interesting discussion of the role of the county society in combating the new King-Anderson Bill, HR 3920. He finished by showing a 14-minute film on the subject by Edward R. Annis, M.D., President of the American Medical Association.

The Cumberland County Medical Society met at the Eastland Motor Hotel in Portland, Maine on November 21, 1963. Seventy-three members and guests were present. After a social hour and dinner, the meeting was called to order by the President, Philip P. Thompson, Jr., M.D.

The obituaries of Drs. Henry W. Hanson, Jr. and Carol Schwartz were read and it was voted that these be spread on the records of the society and that copies be sent to the families.

A nominating committee, consisting of Maurice Van Lonkhuyzen, M.D., Chairman, Philip S. Fogg, Jr., M.D. and George F. Sager, M.D., was appointed by the President to present a slate of officers at the annual meeting.

The remainder of the evening was devoted to a talk on American Medical Political Action Committee by Mr. Edward Donelan, the New England Field Representative of this organization.

The annual meeting of the Cumberland County Medical Society was held on December 19, 1963 at the Eastland Motor

Hotel in Portland, Maine. Seventy-eight members and guests were present. After the social hour and dinner, the meeting was called to order by the President, Philip P. Thompson, Jr., M.D.

Howard P. Sawyer, Jr., M.D., of the Legislative Committee, reported that hearings of the King-Anderson Bill had been held up in committee after the death of President Kennedy and would be reopened for hearing for four days in January. He urged the membership to write to their congressmen and members of the House Rules Committee expressing their opposition to this bill.

It was voted that C. Eugene Fogg, M.D. and Thomas A. Foster, M.D. be recommended for Honorary membership in the Maine Medical Association and the Cumberland County Medical Society and that Drs. William L. Casey, William E. Freeman, Herman C. Petterson and Daniel M. Rowe be recommended for Senior membership. It was voted that Dr. Marion W. Westermeyer be voted to Junior membership for the duration of his residency in anesthesia at the Maine Medical Center.

Maurice Van Lonkhuyzen, M.D., chairman of the nominating committee, presented a slate of officers for 1964 and the entire slate, which follows, was approved:

President, Charles R. Geer, M.D., Portland  
Vice-President, Richard J. Goduti, M.D., Portland  
Secretary-Treasurer, Stanley B. Sylvester, M.D., Portland  
Public Relations Committee: Eugene C. McCann, M.D.,  
Chairman; Alvin A. Morrison, M.D. and Laban W. Leiter,  
M.D., all of Portland

Delegates to the Maine Medical Association House of Delegates: Drs. Robinson L. Bidwell, Donald P. Cole, John F. Gibbons, Charles R. Glassmire, Merle S. Bacastow, Louis G. Bove, Philip S. Fogg, Jr., Howard P. Sawyer, Jr., Philip P. Thompson, Jr., Maurice Van Lonkhuyzen, Stanley B. Sylvester. Alternates: Lloyd G. Davies, Stanley W. Kent, A. Dewey Richards, George F. Sager, Clifford W. Gates, Clement A. Hiebert, Stephen E. Monaghan, Hugh P. Robinson, Howard M. Sapiro, William J. Tetreau.

Dr. Thompson reviewed the activities of the county society during the past year, outlining first the objectives to the King-Anderson Bill, which should be used by the membership in explaining the stand of organized medicine in opposition to this bill.

(1) First, its excess cost to the same coverage could be obtained from Blue Cross-Blue Shield at one-half or even a third the cost.

(2) Hospitals now overcrowded will be swamped by the elderly, many of whom will not need hospitalization.

(3) Fifty-six percent of those over 65 now have health insurance.

He announced that the United States Public Health Service is studying hepatitis in Maine and particularly desires blood from patients who have developed hepatitis. This can be obtained if such patients have given blood transfusions or donated blood shortly before coming down with hepatitis.

He stated that the Legislative Committee has been very active and should be continued at the county level and that the so-called steering committee, consisting of the officers, delegates, chairman of committees, and councilors have met several times in the past year; and this gives promise of being a very useful committee.

Several other items of community interest were discussed; particularly the fact that on several occasions the medical society allowed a group to embark on various campaigns involving medical care and public health problems. Examples of

this are the fluoridation problem, oral polio unification attempts of the United Community agencies and organization of nursing homes for chronic care. He proposed that a committee of the society should be formed to attempt to coordinate these activities and channel them in the proper direction.

A musical interlude closed the meeting. This was provided by the efforts of three distinguished trumpeters, to wit, Drs. Clement A. Hiebert, John A. Godsoe and Mr. Raymond Lebel.

ALBERT ARANSON, M.D.  
*Secretary*

#### SOMERSET

A meeting of the Somerset County Medical Society was held at the Oak Pond Restaurant in Canaan, Maine on October 15, 1963.

Richard P. Laney, M.D. introduced the guest speaker, John P. Dow, M.D. who has recently settled in Pittsfield, Maine. Dr. Dow presented an interesting paper on the more Recent Advances in Medicine, referring especially to the newer laboratory tests which confront the physician of today.

Alan T. Webb, M.D., Chief Pathologist and head of the laboratory at the Rancho Los Amigos in Los Angeles, California was also a guest speaker. The Rancho Los Amigos, a 2100 bed hospital, deals mainly with the care of the chronically ill and the rehabilitation of these cases. Dr. Webb showed colored slides of the physical plant at this county institution, describing some of the work carried on there.

MARIAN L. STRICKLAND, M.D.  
*Secretary*

#### LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on November 19, 1963. Guests present were Ernest W. Stein, M.D., President of the Maine Medical Association and Eugene C. McCann, M.D. of Portland.

Dr. Stein extended greetings from the Maine Medical Association and Francis A. Winchenbach, M.D. mentioned programs of interest to physicians which are being planned for ETV.

Dr. McCann spoke on The Use of Medications and The Management of Pregnancy.

The regular monthly meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on December 17, 1963.

A letter from Edward R. Annis, M.D., President of the American Medical Association, was read alerting the membership to the need to foment lay opposition to King-Anderson legislation.

Emerson H. Drake, M.D., of Portland, spoke on Recent Advances in Cardiac Surgery.

GEORGE W. BOSTWICK, M.D.  
*Secretary*

#### PENOBSCOT

A meeting of the Penobscot County Medical Society was held at the Millinocket Community Hospital in Millinocket, Maine on November 19, 1963. Thirty-five members were present with Allison K. Hill, M.D., President, presiding.

Reports of the Public Relations Committee were read. TV programs, sponsored by the society, are scheduled for the winter months. The committee suggested that every member read the King-Anderson Bill, HR 3920.

The polio committee reported that the third and last county-wide clinic had been held on November 17th, during which 50,000 doses of type 3 oral polio vaccine were administered. The committee thanked the members for their help in this program.

Resolutions were read on the death of Watson S. Purinton, M.D.

The guest speaker, Allan D. Callow, M.D., of the New England Center Hospital, spoke on Surgical Considerations in Cerebral Vascular Insufficiency. He described the technique of diagnosis and therapy and presented his results in a series of patients.

The annual meeting of the Penobscot County Medical Society was held at the Tarratine Club in Bangor, Maine on December 17, 1963. The President, Allison K. Hill, M.D., presided.

The speaker of the evening was Mr. Robert C. Hawkes, Director of the Speech and Hearing Center of Bangor, who described the facilities available and the services offered to local patients. He discussed briefly the problem of Aphasia; its definition, history in medical literature and described various tests for examination and diagnosis of this affliction.

The following officers were elected for 1964:

President, William A. Purinton, M.D., Bangor  
Vice-President, Richard T. Munce, M.D., Bangor  
Secretary, Hadley Parrot, M.D., Bangor  
Treasurer, Benjamin L. Shapero, M.D., Bangor  
Executive Council, Robert O. Kellogg, M.D., Bangor

Dr. Hill expressed his appreciation to the members for their service on committees, and for their cooperation with the officers during the year.

FREDERICK C. EMERY, M.D.  
*Secretary*

#### KENNEBEC

A meeting of the Kennebec County Medical Association was held at the Veterans Administration Center in Togus, Maine on November 21, 1963.

A nominating committee was appointed by Brinton T. Darlington, M.D. to present a slate of officers at the annual meeting in December for the year 1964.

Guy W. Leadbetter, M.D., of the Massachusetts General Hospital, presented the clinical portion of the program, speaking on Recent Developments in Urinary Tract Abnormalities, Observations and Infection; Their Prognosis, Evaluation and Treatment.

The annual meeting of the Kennebec County Medical Association was held on December 12, 1963 at the Augusta State Hospital in Augusta, Maine.

The following officers were elected for the year 1964:

President, Kenneth W. Sewall, M.D., Waterville  
Vice-President, John D. Denison, M.D., Gardiner  
Secretary-Treasurer, Earle M. Davis, M.D., Waterville  
Delegates to the Maine Medical Association House of Delegates: Brinton T. Darlington, M.D., Lane Giddings, M.D. and Napoleon J. Gingras, M.D., all of Augusta; John D. Denison, M.D., Gardiner and Samson Fisher, M.D., Waterville. Alternates: Oakley A. Melendy, M.D. and William N. Runyon, M.D., both of Augusta; Richard E. Barron, M.D., Monmouth; Irving I. Goodof, M.D. and Paul A. Jones, Jr., M.D., both of Waterville  
Councilors: Allan J. Stinchfield, M.D., Augusta (3 yrs.); Robert L. Ohler, M.D., Togus (2 yrs.) and Richard H. Dennis, M.D., Waterville (1 yr.)  
Grievance Committee: John F. Reynolds, M.D., Chairman,



Waterville; Oakley A. Melendy, M.D., Augusta and George I. Gould, M.D., Richmond

Man Power Procurement and Civil Defense Committee: Frank B. Bull, M.D., Chairman, Gardiner; Albert A. Poulin, M.D., Waterville and M. Tieche Shelton, M.D., Augusta

John C. Patterson, M.D., Superintendent of the Augusta State Hospital, spoke on The Changing Role of the State Hospital.

EARLE M. DAVIS, M.D.  
*Secretary*

## HANCOCK

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on December 11, 1963.

The following officers were elected for the coming year: President, Elizabeth E. Williamson, M.D., Blue Hill Vice-President, Russell G. Williamson, M.D., Blue Hill Secretary-Treasurer, Frank S. Cruickshank, Jr., M.D., Bar Harbor

Delegates to the Maine Medical Association House of Delegates: Russell M. Lane, M.D., Blue Hill and Llewellyn W. Cooper, M.D., Bar Harbor. Alternates: Herbert T. Wilbur, Jr., M.D., Southwest Harbor and Philip L. Gray, M.D., Blue Hill

Board of Censors: W. Edward Thegen, M.D. (3 yrs.) and Arthur M. Joost, Jr., M.D. (2 yrs.), both of Bucksport and Robert F. Russell, M.D., Penobscot (1 yr.)

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## WASHINGTON

A regular meeting of the Washington County Medical Society, in conjunction with the St. Croix Medical Society, was held at the Charlotte County Hospital in St. Stephen, New Brunswick, Canada on December 12, 1963. Fifteen members and two guests were present.

Roy Creamer, M.D. of St. John, New Brunswick, Canada spoke on Postpartum Hemorrhage.

A meeting of the Washington County Medical Society followed with the President, James C. Bates, M.D., presiding.

The following officers were elected for 1964:

President, James C. Bates, M.D., Eastport

Vice-President, Robert G. MacBride, M.D., Lubec

Secretary-Treasurer, Karl V. Larson, M.D., East Machias

Board of Censors: DaCosta F. Bennet, M.D., Lubec (3 yrs.)

A meeting of the St. Croix Medical Society was then held with remarks by Hazen C. Mitchell, M.D., on the Executive Board of the New Brunswick Medical Society.

KARL V. LARSON, M.D.  
*Secretary*

## New Members

### ANDROSCOGGIN

Dorothy Anderson, M.D., 369 Main St., Lewiston

### CUMBERLAND

Anthony Betts, M.D., Maine Medical Center, Portland (By transfer from Lincoln-Sagadahoc County Medical Society)

James L. Fife, M.D., Baribeau Dr., Brunswick

Stephen J. Kurzbard, M.D., AFES, 500 Forest Ave., Portland

### FRANKLIN

Paul A. Brinkman, M.D., Farmington

### KENNEBEC

Leandre W. Giguere, M.D., 30 Elm St., Waterville

Bruce Trembly, M.D., 33 College Ave., Waterville

### LINCOLN-SAGADAHOC

Nelson P. Blackburn, M.D., Bath Memorial Hospital, Bath (By transfer from Penobscot County Medical Society)

John J. McLaren, M.D., Baribeau Dr., Brunswick

### PENOBSCOT

John C. Bjorn, M.D., Hampden Highlands

George O. Chase, M.D., Eastern Maine General Hospital, Bangor (By transfer from Cumberland County Medical Society)

John F. McGinn, M.D., 205 French St., Bangor

### PISCATAQUIS

Felix M. Garcia-Rey, M.D., 14 Charles St., Milo

Araminta Rodriguez, M.D., 14 Charles St., Milo

### WASHINGTON

George B. Shaw, M.D., Main St., Jonesport

# News, Notes and Announcements

**State of Maine Board of Registration of Medicine**  
**Secretary — George E. Sullivan, M.D.**  
**Waterville, Maine**

**Physicians Licensed to Practice Medicine and**  
**Surgery in the State of Maine**  
**November 12-14, 1963**

## THROUGH EXAMINATION

Eliseo N. Aiello, M.D., The Hospital of St. Raphael, New Haven, Conn.

Ahmet M. Aytur, M.D., St. Mary's Hospital, Cincinnati, Ohio

Anthony B. Browne, M.D., Mass. General Hospital, Boston, Mass.

Christopher P. Dimitriadis, M.D., Lowell General Hospital, Lowell, Mass.

Dimitrios Dimitriou, M.D., 306½ S. Main St., Suffolk, Va.

Ho S. P. Dinesoy, M.D., Howard University, Washington, D. C.

Hans H. Epstein, M.D., Woodstock Medical-Dental Centre, Woodstock, N. B.

Edmundo A. Kauffmann, M.D., 270 E. 45, Brooklyn, N. Y.

Shamsher A. K. Khera, M.D., 7745 Sherbrooke E., Montreal, Quebec, Can.

J. Odin Maldonado, M.D., 4875 Sherbrooke St., Westmount, Montreal, Can.

Avelino M. Mape, M.D., Utica State Hospital, Utica, N. Y.

Ulrich Moeser, M.D., 20 Kenilworth St., Portland, Me.

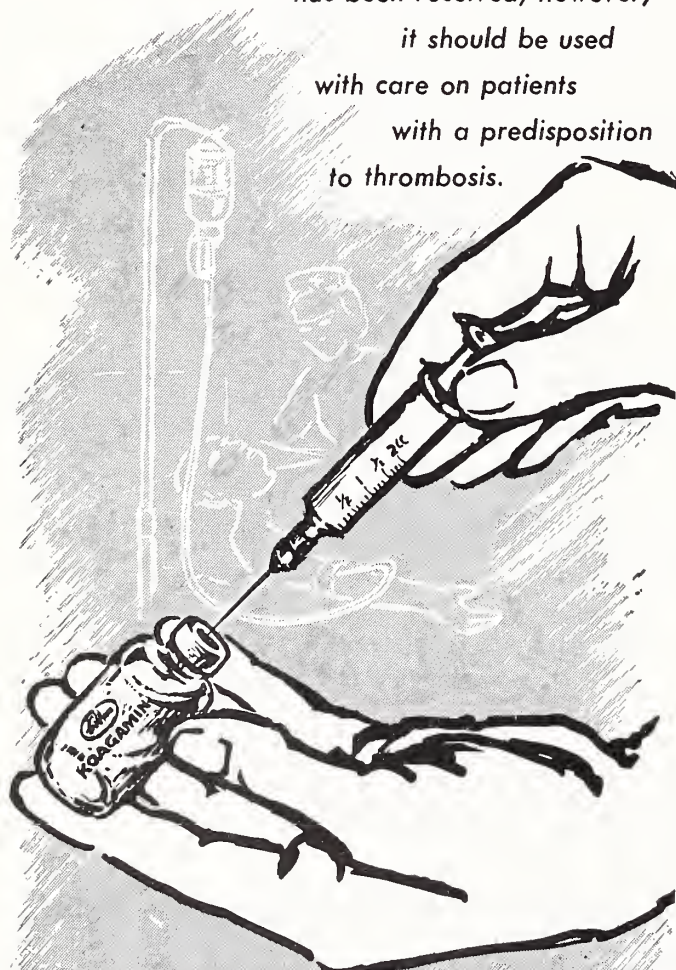
Ken Mori, M.D., Maimonides Hospital of Brooklyn, Brooklyn, N. Y.

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Manuel G. Pena, M.D., 74 McClane Ave., Washington, Pa.  
Francisco E. Pflaum, M.D., Box C, Pownal, Me.  
Carlos R. S. Santana, M.D., 311 Fairview Ave., Fairview, N. J.  
Eugene Toker, M.D., West Brentwood, L. I., N. Y.  
Ignatios J. Voudoukis, M.D., Maine Medical Center, Portland, Me.  
Samuel D. J. Yeh, M.D., 303 East 71st St., New York, N. Y.  
Daniel A. Zelling, M.D., 747 Carnegie Ave., Akron, Ohio

### THROUGH RECIPROCITY

Achsa M. Bean, M.D., Star Route 2282, Owl's Head, Me.  
Tracy D. Cuttle, M.D., Portsmouth Naval Shipyard, Portsmouth, N. H.  
Richard C. Dillihunt, M.D., Maine Medical Center, Portland, Me.  
Stephen J. Kurzbard, M.D., 24 Forest Pk., Portland, Me.  
Alfred F. Lagace, M.D., Cary Memorial Hospital, Caribou, Me.  
Murray A. Leavitt, M.D., 173 Garland St., Bangor, Me.  
Richard A. Levy, M.D., Maine Medical Center, Portland, Me.  
William D. McLarn, M.D., Maine Coast Memorial Hospital, Ellsworth, Me.  
John R. Niceforo, M.D., 104 Merrymeeting Rd., Brunswick, Me.  
William A. O'Brien, M.D., Arthur R. Gould Memorial Hospital, Presque Isle, Me.  
Daniel N. Slatkin, M.D., Box 488, Montefiore Hospital, New York, N. Y.  
Edward Tober, M.D., 1292 Ocean Blvd., Rye, N. H.  
Joseph Winsten, M.D., 43 Fair Oaks Dr., Lexington, Mass.

### REPORT OF DELEGATE — Continued from page 10

medical societies, religious groups and other related organizations;

Received a report on the AMA Members *Retirement Plan* and urged physicians to act quickly if they are to exercise their rights under Public Law 87-792 during 1963;

Asked the Association staff to study the feasibility of opening the *Clinical Meeting* two Sundays prior to Thanksgiving Day;

Suggested that an appropriate committee of the AMA work with the United States Public Health Service and the industry in providing a type of *detergent* that will assure safety to the health of the public;

Urged all AMA members to continue to support the *Woman's Auxiliary* so that it can be successful in increasing its membership, raising more revenue and broadening its range of activities.

Merck Sharp and Dohme pharmaceutical company made its third contribution of \$100,000 to the student loan fund of the American Medical Association Education and Research Foundation. The AMA-ERF also received a total of almost \$400,000 from physicians in three states for financial aid to medical schools.

This Clinical Meeting of the AMA which was held in Portland, Oregon, was attended by:

Dr. Ernest W. Stein, President of the MMA; Dr. Clyde I. Swett as delegate of the Department of General Surgery, Dr. Paul H. Pfeiffer, alternate delegate of the MMA, and myself.

ASA C. ADAMS, M.D.  
Delegate to AMA



Officers of the Maine Medical Association — 1963-64

*President*, ERNEST W. STEIN, M.D., Pittsfield  
*President-elect*, THOMAS A. MARTIN, M.D., Portland  
*Speaker of the House of Delegates*, LINUS J. STITHAM, M.D., Dover-Foxcroft

<i>Councilors</i>	<i>Term Expires</i>	<i>Councilors</i>	<i>Term Expires</i>
First District	1966	Sixth District	1964
PAUL S. HILL, JR., M.D., Saco		CLYDE I. SWETT, M.D., Island Falls	
Second District	1966	Immediate Past President	1964
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Third District	1965	Delegate to AMA	Jan. 1, 1965
JOHN F. DOUGHERTY, M.D., Bath, Chm.		ASA C. ADAMS, M.D., Orono	
Fourth District	1965	Alternate Delegate to the AMA	Jan. 1 1965
GEORGE E. SULLIVAN, M.D., Fairfield		PAUL H. PFEIFFER, M.D., Waterville	
Fifth District	1964	Executive Director	
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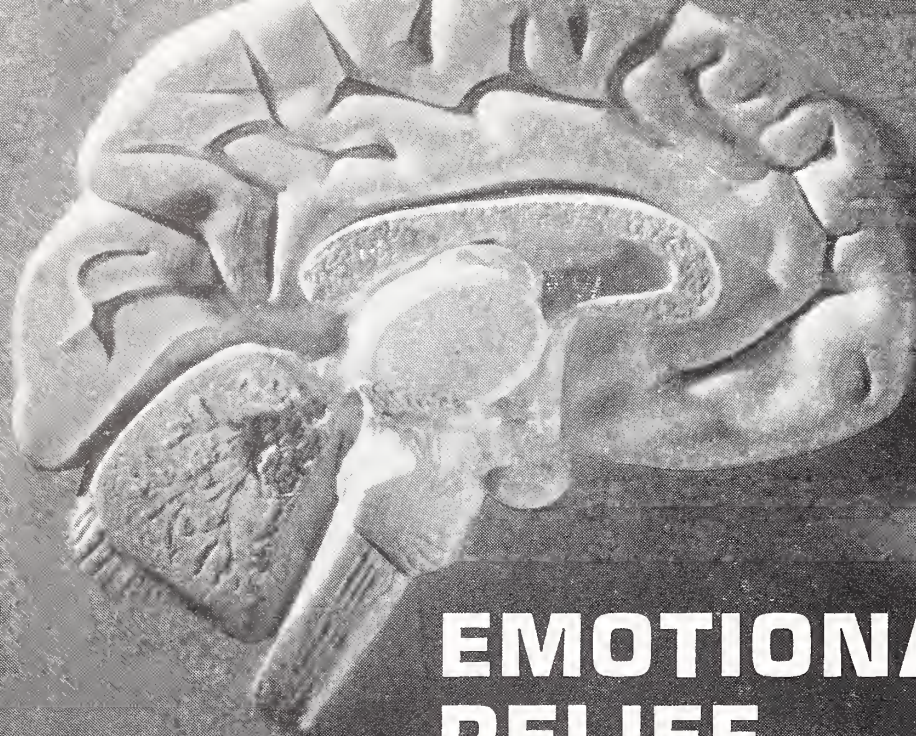
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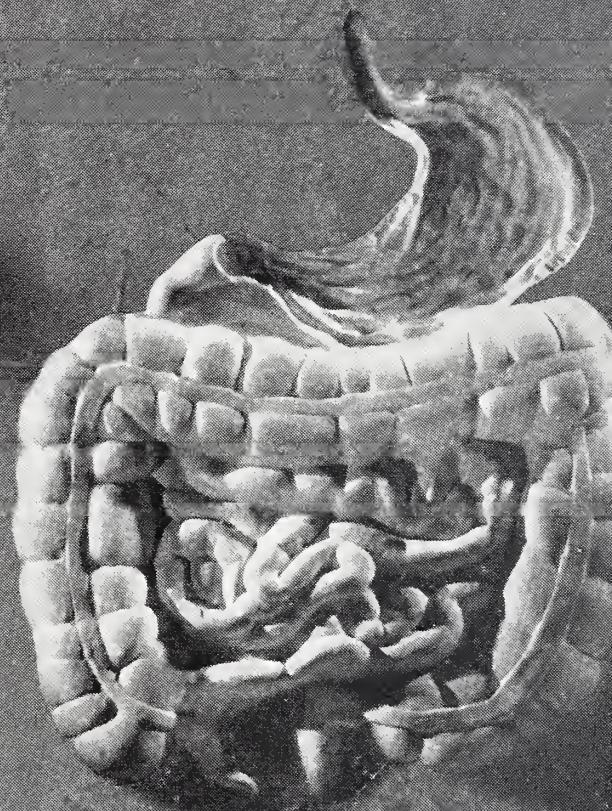
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# The Journal of the Maine Medical Association

Volume Fifty-Five

Brunswick, Maine, February, 1964

No. 2

## Care Of The Child With An Enduring Disability\*

Ideas And Aspects Of Services Utilizing Resources Of A Rural  
Maine Community Medical Center

EDMUND N. ERVIN, M.D.\*\*

Several years ago in response to an urgent need for services for mentally retarded children, the Congress of the United States allocated funds to the Children's Bureau for programs in the various states. The need for diagnostic and evaluation services for these children in Maine was urgent. The State Legislature in 1955 had passed an amendment to Law-Chapter 467, Sec. 1, R.S.C. 41, paragraphs 207A-I, providing subsidy for classes for educable mentally retarded children, those with an I. Q. between 50-75, through the Department of Education. There was no service providing diagnosis, counseling of parents regarding prognosis, training and management, or treatment of associated handicaps. It was felt that such a service for the mentally retarded pre-school child was important at the earliest possible age to make and confirm the diagnosis, to offer a constructive plan of management so that such children might develop to the fullest extent of their abilities, and to avoid the emotional consequences which might result to both child and family from unwise and inept counseling. A clinic for mentally retarded pre-school children was opened at the Thayer Hospital, Waterville, Maine, in 1957 under the auspices of the Division of Maternal and Child Health and Crippled Children Services of the State Department of Health and Welfare. Such a clinic was in accord with the newer philosophy that when needed services are offered to the re-

tarded living at home in the community, the cost to the taxpayer is small, the child develops more fully and the emotional stability of the family group may be preserved.

The clinic was organized with the concept that the child with a handicap, particularly the mentally retarded, was more likely to be multiply handicapped and therefore various medical and para-medical services would be needed in understanding the whole child and the total family situation. Such a clinic would need to involve the efforts of many disciplines if we were to offer worthwhile and comprehensive service. Consequently the staff of the clinic includes (1) Pediatrician as director; (2) Clinical Psychologist; (3) Psychiatric social worker; (4) Speech consultant; (5) Nutritionist; (6) Public Health Nurse; (7) Psychiatric and other medical consultative services. Laboratory and x-ray, home visits by the social worker and the public health nurse, hospitalization whenever necessary, as well as continuing speech therapy, case work, and psychotherapy, whenever indicated, were to be offered.

Each child to be evaluated is seen by every one of the clinic personnel who formulates his own impression of the child. When the workup is completed, the reports and impressions of the staff personnel are thoroughly discussed; (1) in an attempt to make as exact an etiological diagnosis and comprehensive evaluation as is possible within the limits of our abilities and facilities; (2) so that we may understand the numerous factors which may be involved in the problems pre-

\*Presented at the New England Pediatric Society, May, 1963.

\*\*Chief of Pediatrics, Thayer Hospital, Waterville, Maine.

sented as well as the various family and community resources which can be helpful; (3) in order that the recommendations which are made to the family regarding training and management will not be isolated and impractical suggestions but comprehensive and constructive proposals which have considered the whole child, the total family situation and the cultural patterns which exist in Maine today. These are interpreted for the parents in a counseling session, at which time their questions are answered which will lead to greater understanding and acceptance of present and possible future problems.

How do you measure the quality, the effectiveness, the value of such a service? It would be difficult, if not impossible to determine our diagnostic batting average. Unfortunately there is a large group of cases associated with diseases and conditions which we ascribed to an unknown pre-natal influence. This is due in part to our inability to categorize certain findings as pre-natal in origin and to determine the etiological significance of disturbances occurring before or at birth. Some diagnoses might be changed with more definitive neurological studies whereas others will never be diagnosed with certainty until post-mortem histo-pathological examinations are done. It is difficult, if not impossible to evaluate complications of pregnancy partly because the precise knowledge of their effect is not known, the passage of time makes the recollection of significant details by the mother vague and indefinite and because hospital labor and birth records are all too often wanting, except in observation of major complications. The exact nature and etiology of each case of mental retardation depends upon more detailed studies than we are able to do. Each case presents a diagnostic challenge but is it important to the function of this clinic that we know whether the difficulties are the result of damage to or an absence of brain substance if such information does not consider the emotional consequences or influence the therapeutic approach? We need to be able to differentiate progressive neurological disease from the static cases of brain damage, inherited manifestations from their phenotype because of the importance of genetic counseling, major psychoses from personality disturbances which produce a pseudo-retardation and for whom there is a difference in the direction of therapy and lastly to separate those cases who function and test in a sub-normal manner because they have been deprived socially and culturally but who have adequate intellectual equipment from those who are brain-deficient.

It seems to us that making an etiological diagnosis is but one phase of this activity and the value of such a clinic does not depend upon our skill in this area but is to be compared with our effectiveness in counseling and management. Services for the handicapped must be comprehensive enough to evaluate the whole problem, its effect on the child and the family, and to offer a practical program of care and training. Most families are not interested in the why, but in what do we do

now. For this type of guidance the medical profession has been of little help. We could list a score of reasons for this but principally such an effort is beyond the scope and interest of most physicians. These problems need a broader look from many angles and this is the merit of a multidisciplinary clinic. Therefore the recommendations of the para-medical services which are represented in this team approach are important and indispensable. With them we can more nearly put the puzzle together, offer some solution of present problems, and give the family a glimpse into the future. In consideration of the family situation and the community resources we can suggest long-term planning and management within limits which may be less dismal than the future which was imagined.

The emotional problems which overlay the basic one of mental retardation or other handicap often escape detection and certainly solution without several sources of ideas and the combined effort of many disciplines makes this a necessary feature of such a clinic. We see the child more fully. We see the child who is mentally retarded rather than the mentally retarded child. We note the effect of this child on the whole family. Without this information our recommendations may well be meaningless and impractical. In addition, this type of evaluation gives the family an assurance of our interest, concern, and thoroughness which must be the basis for a continuing relationship.

We need, for example, more than a numerical estimate of intelligence. We must know something of the child's abilities as well as his deficiencies and how these relate to his present patterns of behavior. It is important that the family understand these characteristics to be able to interpret his desires and to be consistent in any plan of discipline and training. They must appreciate realistically those functions which will improve little or not at all, thus conserving their energies for the development of the others in which there is more ability and potential. This concept offers hope of progress which, however small and insignificant to the clinician, is important to the emotional stability of the family group. Consequently, the comprehensive evaluation is important therapeutically as well as diagnostically.

Finally, continuity of care and interest must be a feature of these clinic services as it should be for all patient services. Support is given through visits by the public health nurse in their area, continuing speech therapy, and further consultations by the various medical specialties for associated handicaps which often suffices to dispel the threat of institutional placement. If this becomes necessary or desirable we can explain the various aspects of residential care as it is in Maine today.

In these past six years while we've gained in our knowledge of the problems of the mentally retarded it has occurred to us that if such services are of value to this type of handicap, could such comprehensive care be a more satisfactory solution for the problems of



children with other handicaps? If this is so, who should be responsible for the initiation of such services and the continuity of such care? How do you create an awareness of the various aspects and effects of a handicap among physicians, the community, and the public and private agencies?

We have said that the needs and problems of the child with a handicap are multiple and complicated. To meet them, highly specialized clinic services have been developed throughout the country. In the State of Maine, these are often inaccessible, inadequate, or non-existent. Where such services exist they frequently do only a partial job because they often miss in the evaluation and understanding of the whole child and the total family situation. How much time and money, both public and private are consumed with these efforts? There are numerous examples of fragmentation, duplication, and delay in the provisions of services. There is a need for integration of these services into one facility with the use of many disciplines to contribute to comprehensive diagnostic evaluations. It would seem that the purpose and structure of a clinic for one type of handicap, i.e., the mentally retarded, could be a pattern for a service for children with other handicaps. This is not a novel idea, for clinics with a similar philosophy are scattered over the country in Louisiana, Washington, Kansas, Maryland, and South Carolina to name a few. To fulfill this need in the State of Maine and in a somewhat different setting than exists elsewhere, the Mansfield Clinic of the Thayer Hospital, a small medical center in a predominately rural area, has started a multi-purpose evaluation clinic solely for these children.

The clinic will evaluate the mentally retarded, the slow learner, the brain-injured, the partially sighted, the emotionally disturbed, the psychotic, the epileptic, the physically handicapped, the child with speech and hearing problems, or the one who is handicapped as a result of chronic illness. We will follow the procedure as outlined for the clinic for the mentally retarded. The following are examples:

- The cerebral palsied child, who at the age of ten, cannot walk or talk and is found to have a non-verbal level of intelligence in the dull normal range.
- The hard of hearing child who is classified as mildly retarded by psychological data but correctly diagnosed and helped is found to test in the range of above average intelligence.
- The "brain damaged" child with an I. Q. of over 100 who cannot participate in regular classes because of emotional lability and difficulty with reading and number concepts.
- The psychotic child whose intellectual functioning is impaired.

- The child who has emotional problems because of a chronic illness of long standing and who presents handicaps for learning because of his frequent and persistent absence from school.
- The child with epilepsy whose family and teachers need an understanding of the nature and manifestations of his illness.

New ideas of medical care for the child with a handicap will evolve. These will vary depending upon the location of the facility. They will be somewhat different in Maine spread thinly as we are over a wide geographic area. Separated by some distance from the clinic, will such services be adequate and give the continuity of care and interest which will be essential to good management and some progress? Can we circumvent long-term hospitalization or institutional care? Most parents want the child at home. For the retarded residential facilities are available for only about 5-10% of the total number, therefore the great majority are scattered through isolated communities. We have no idea of the particular problems which we will encounter. However, many children have reached a dead-end for lack of any kind of service because in this predominantly rural area, some of these communities have little or no medical service and no other resources which can offer support. There needs to be a central agency with a philosophy of continuing service and interest in all areas of the child's activities which will do this.

As important as the clinic facility will be, one of the key figures must be the referring physician. Referrals will be accepted from physicians only. This means a considerable educational effort. He must appreciate that children are different, the significance of these variations, and that these may need investigation. The absence of adequate facilities has created among physicians an attitude of futility. He will need to know that some of his questions can be answered and these he can transmit to the families. We need to make him feel that he alone is not responsible but there is an agency close at hand who is willing to share his concern. The calibre of the reports which go out to him will do some of this but he will be invited to the post-clinic sessions when his particular case is finally discussed. An effective liaison between the clinic and the physician will be an important educational feature for the clinic personnel as well, in that it keeps the problems of the children continuously before them and will make their recommendations practical and knowledgeable. Finally the community and any other agencies need to be involved and to feel a sense of responsibility, for school planning and social adjustment in the community will be as important as the prevention of problems by early identification and evaluation.

2 School Street, Waterville, Maine

# Internal Carotid Aneurysms

## "A Different Breed Of Cat"

BRUCE TREMBLY, M.D.

Aneurysms of the intracranial portion of the internal carotid artery may present a clinical picture that differs from that seen with cerebral aneurysms in other locations. Increasing numbers of internal carotid aneurysms are being detected prior to actual rupture because of physicians' awareness of the significance of early symptoms. In addition, these aneurysms are among the most favorable from the standpoint of successful treatment, and their overall mortality rate is lower than that of most other intracranial aneurysms. In various series, these aneurysms constitute 30%-45% of all intracranial aneurysms. It is the aim of this paper to emphasize certain distinguishing clinical features of internal carotid aneurysms as seen in a large series of patients from Hartford (Connecticut) Hospital. Symptoms, signs, operations and outcome in 71 patients with internal carotid aneurysms are compared with those of 120 patients with intracranial aneurysms in other locations.

### SOURCE MATERIAL

The 216 patients in this series were admitted to Hartford Hospital between the years 1949 and 1963. All but 6 of these patients were under the care of members of the Department of Neurosurgery and its resident staff.\* All of these patients had intracranial aneurysms proven by arteriography (192 patients) or autopsy (25 patients). Patients with subarachnoid hemorrhage without demonstrable aneurysms, and patients dying from other causes in whom aneurysms were incidentally discovered were excluded from this series. In each instance the patient's aneurysm was considered to be the source of symptoms.

There were 25 patients with multiple demonstrated aneurysms. In many of these patients it was difficult to determine from the clinical and arteriographic picture which of 2 or more aneurysms had ruptured. Some were treated conservatively because of bilateral aneurysms at several locations. In those undergoing craniotomy, the operative mortality (75%) was greater than in patients with a single aneurysm. Nine of these patients had aneurysms of the internal carotid artery as well as of other arteries and would thus be included in both groups under comparison. Therefore, for the sake of simplicity, all 25 patients with multiple aneurysms are excluded from tabulation of symptoms, signs and outcome. Distribution of aneurysms in these patients is indicated in

TABLE 1

ANATOMIC DISTRIBUTION OF 247 INTRACRANIAL ANEURYSMS IN 216 PATIENTS				
	(A) 191 Single Aneurysms (191 Patients)		(B) 56 Multiple Aneurysms (25 Patients)	
	No.	Percentage of 191	No.	Percentage of 56
Internal Carotid	71	37%	18	32%
Anterior Cerebral	9	5%	3	5%
Anterior Communicating	46	24%	10	18%
Proximal Middle Cerebral	18	9%	10	18%
Middle Cerebral Trifurcation	29	15%	8	14%
Posterior Communicating	5	3%	4	7%
Posterior Cerebral	3	2%	1	2%
Posterior Fossa (Basilar, Vertebral, Posterior Inferior Cerebellar)	9	5%	2	4%
Choroid Plexus	1	0.5%	—	—
	191	100%	56	100%

TABLE 2

AGE DISTRIBUTION BY DECADES* INTERNAL CAROTID COMPARED WITH ALL OTHER ANEURYSMS 191 PATIENTS WITH SINGLE ANEURYSMS							
Age	Internal Carotid Aneurysms			All Other Aneurysms			Total
	Male	Female	Subtotal	Male	Female	Subtotal	
0-9	0	0	0	0	1	1	1
10-19	2	1	3	1	0	1	4
20-29	1	4	5	4	4	8	13
30-39	6	9	15	18	10	28	43
40-49	5	12	17	19	23	42	59
50-59	2	17	19	15	11	26	45
60-69	2	8	10	6	5	11	21
70-79	0	2	2	0	2	2	4
80—	0	0	0	0	1	1	1
Totals	18	53	71	63	57	120	191

\* Age at time diagnosis was made.

Table 1(B). The remaining 191 cases were otherwise unselected.

### INCIDENCE

Aneurysms of the internal carotid artery comprise the largest single group in the present series (Table 1). There is minimal difference between the 2 groups with respect to age distribution (Table 2), but there is an impressively high ratio of females to males in patients with internal carotid aneurysms (Table 3).

\*The 6 exceptions were examined by a neurosurgeon but died on other services before transfer could be accomplished.



TABLE 3

SEX DISTRIBUTION IN INTERNAL CAROTID COMPARED WITH ALL OTHER ANEURYSMS 191 PATIENTS WITH SINGLE ANEURYSMS				
	Male	Female	Total	Female/Male Ratio
Aneurysms of Internal Carotid Artery	18	53	71	2.94:1
Aneurysms of All Other Intracranial Arteries	63	57	120	0.95:1
Total	81	110	191	1.36:1

ONSET

The onset of symptoms was gradual in 14 patients (20%) with internal carotid aneurysms (Table 4). These symptoms included: gradually increasing headache, retro-orbital or facial pain, blurred or double vision, drooping of eyelid, and dizziness. In the remaining 57 patients in this group, symptoms came on suddenly and catastrophically. In 6 patients (8%) the onset was associated with physical stress (lifting, bending, intercourse). In contrast, only 1 of 120 patients with aneurysms in other locations had gradual onset of symptoms. Almost twice as many (15%) were associated with stress. Both groups had a similar incidence of unusual symptoms preceding the sudden onset by several days or weeks. These symptoms were not considered to have been due to previous rupture, and included an unusual degree of headache, eye pain, dizziness, blurred vision, double vision, nausea and unilateral weakness.

ADMISSION EXAMINATION

Findings on admission are indicated in Table 5. Most patients were admitted directly to Hartford Hospital, and detailed examination was made within a few hours of onset of symptoms. In patients first admitted to other hospitals and later transferred to Hartford Hospital, the findings on admission to the first hospital are included here.

Consciousness was not impaired in 43 (61%) patients with internal carotid aneurysms. This means that the patient was awake and alert and able to give an adequate history. Of patients with internal carotid aneurysms, all those with gradual onset of symptoms were awake and alert at the time of admission to the hospital. Increased blood pressure (greater than 130 mm. systolic) did not appear to correlate with any other findings on admission. Of those patients awake and alert at the time of admission, half had ocular signs as compared with only one fourth of the group with impaired consciousness. Ocular signs included dilatation of pupil, varying degrees of ophthalmoplegia, and ptosis. Paresis was not usually associated with ocular signs, and most of the patients with paresis had some degree of impaired consciousness.

Of 71 patients with internal carotid aneurysms, 16 were thought not to have bled (Table 5). All of these

TABLE 4

ONSET AND PRESENTING SYMPTOMS IN 191 PATIENTS WITH SINGLE ANEURYSMS				
	(A) Internal Carotid Aneurysms		(B) All Other Aneurysms	
	No.	Percentage of 71 Patients	No.	Percentage of 120 Patients
Gradual onset	14	20%	1	1%
Associated stress	6	8%	18	15%
Symptoms preceding				
sudden onset	13	18%	20	17%
Headache	61	86%	111	92%
Vomiting	23	32%	49	41%
Convulsion	6	8%	14	12%
Loss of consciousness	25	35%	62	52%
Paresis (weakness, aphasia)	10	14%	19	16%
Visual symptoms	23	32%	5	4%
Behavior changes	1	1%	7	6%

TABLE 5

FINDINGS ON ADMISSION TO HOSPITAL IN 191 PATIENTS WITH SINGLE ANEURYSMS				
	Internal Carotid		All Others	
	No.	Percentage of 71 Patients	No.	Percentage of 120 Patients
Consciousness				
Unimpaired	43	61%	29	24%
Mildly impaired	9	13%	35	29%
Stuporous	9	13%	27	22%
Comatose	10	14%	28*	23%
Stiff Neck	23	32%	49	41%
Paresis	20	28%	39	32%
Ocular signs	32	45%	9	7%
Blood pressure elevated	33	46%	80	67%
Previously documented				
hypertension	15	21%	37	31%
Aneurysm apparently				
not ruptured	16	23%	1	1%
One bleeding episode	43	60%	97	81%
Two or more previous bleeds	12	17%	22	18%

\*One patient's level of consciousness not recorded.

16 patients were awake and alert at the time of admission, and 13 had gradual onset of symptoms. All but 1 of these patients had signs of third or sixth nerve deficit, and the 1 had facial pain with hypesthesia in the distribution of the first and second divisions of the trigeminal nerve. In half these 16 patients, lumbar puncture was done, and in each instance the spinal fluid was clear and under normal pressure. Carotid arteriography was carried out in all these patients.

Forty-three of 71 patients with internal carotid aneurysms had apparently bled only once. All but 1 had sudden onset of symptoms. The patient with gradual onset of symptoms did have bloody spinal fluid. Eighteen of these 43 patients had no impairment of consciousness, and 13 had ocular signs on admission. Of the 5 patients in this subgroup upon whom no lumbar puncture was done, 2 had stiff neck associated with the sudden onset of

severe headache with vomiting and nausea, 2 had evidence of subarachnoid hemorrhage at autopsy soon after admission, and 1 of these was thought to have probably ruptured with severe headache and loss of consciousness.

In contrast, in the group of 120 patients with aneurysms in other locations, only 29 (24%) were awake and alert at time of examination and 28 (23%) were in coma. There was a higher overall incidence of elevated blood pressure and of previously documented hypertension in this group and a much lower incidence of ocular signs.

Only 1 patient in this group was thought not to have bled. This was a man with a middle cerebral trifurcation aneurysm who had sudden onset of aphasia without headache or alteration of consciousness. There was a similar incidence of suspected previous bleeding episodes in the 2 groups.

### OUTCOME

Inspection of Table 6 will show that a much smaller percentage of patients with internal carotid aneurysms died prior to operation as compared with patients in the other group (Section 1). This reflects the larger number of patients in the first group who were awake and in good general condition at the time of admission. Most of those who died prior to operation without evident rehemorrhage did so before the fourth hospital day. Most rehemorrhages occurred after the tenth day.

Operative mortality (Section 2) includes every death during the hospital admission under study. Two patients with internal carotid aneurysms, 1 who underwent 2 craniotomies and 1 who had proximal carotid ligation, died from apparent rehemorrhage shortly after discharge home. They are included in this mortality group. All patients who died after carotid ligation (alone or with craniotomy) had rebled. "Alive with minimal deficit" refers to the patient's condition at the time of discharge from the hospital. These patients were able to care for themselves, to speak, ambulate and cerebrate well. Their return to regular daily activity was expected. The remaining patients (not included in Table 6) had a "substantial" neurologic handicap at the time of discharge, including: hemiparesis, aphasia, severe memory deficit, and impairment of consciousness.

Sections 3 to 7 are subgroups of Section 2. "Optimum" patients were fully awake, stable during the preoperative period, not substantially impaired in neurologic function, and had normal spinal fluid pressure at time of surgery. "Poor-terrible" refers to patients in deep stupor or coma with severe neurologic impairment and with evidence of increased intracranial pressure. Patients falling between the 2 categories are not included

in this tabulation. It is evident that patients in optimal condition at time of operation fared far better than those in poor condition. It can also be seen that proximal carotid ligation has a lower mortality and a higher "good result" rate in patients with internal carotid aneurysms than in patients with all other aneurysms.\*

Section 8 refers to the overall result as far as can be determined from study of office and clinic records. It is beyond the scope of this paper to present detailed analysis of long term results, but a few general statements can be made. Almost half those patients with minimal deficit at time of discharge from the hospital were examined during the year prior to this survey (July, 1963). About one-fourth of those patients with a neurologic handicap were seen during the same period of time.

Of patients in both aneurysm groups with little or no neurologic deficit at discharge, more than 80% were seen in follow-up from 3 months to 9 years after operation. These people continued to do well with gradual resolution of remaining neurologic abnormality. Patients with internal carotid aneurysms commonly had extraocular movement deficits, most of which gradually subsided during the first postoperative year. There were several psychiatric patients in both groups and a few patients with seizures.

Of those patients in both aneurysm groups with "substantial" neurologic deficit, half were not seen again following discharge. Many were sent to nursing homes or chronic care hospitals, and follow-up information is not available. About one-fourth of the total number of these patients had improved to the point of being self-sufficient at the time of last examination. The remainder were still handicapped when last seen.

### COMMENTS

From the information presented here, several features of internal carotid aneurysms appear to distinguish many of them from intracranial aneurysms in other locations.

(a) A large number (23%) of patients in this group were diagnosed and treated without the aneurysm having ruptured. All these patients were alive at last follow-up examination, and only 1 was worse as the result of surgery.\* Poppen and Fager<sup>1</sup> reported that 12 of 101 patients undergoing carotid ligation for aneurysm had not bled. In an autopsy series of 113 intracranial aneurysms, Housepian and Pool<sup>2</sup> reported that 19 (17%) had not ruptured. Five of these were internal carotid aneurysms and 5 were in the posterior fossa. None had been symptomatic.

(b) Twenty percent of patients in this series with internal carotid aneurysms had gradual onset of symptoms over a period of several days or weeks.

(c) All patients in this series whose internal carotid aneurysms apparently had not ruptured had no impair-

\*This procedure was used in 7 patients with aneurysms of the anterior cerebral, anterior communicating, proximal middle cerebral, and posterior communicating arteries. The 1 survivor had an aneurysm of the proximal middle cerebral artery and was in "optimal" condition at time of operation 6 weeks after hemorrhage. Follow-up for 8 years with no recurrence.

\*She was an elderly lady who had undergone a long and difficult craniotomy with subsequent temporal lobe infarction.



TABLE 6

OUTCOME - 191 PATIENTS				
	Internal Carotid Aneurysms		All Other Aneurysms	
	No.	Percentage of 71 Patients	No.	Percentage of 120 Patients
1. Non-operated: Total	12	17%	43	36%
Dead	8	11%	38	32%
Without evident rebleed	7		21	
With evident rebleed	1		17	
Alive	4		5	
2. Operated: Total	59		77	
Dead (operative mortality)	13	22% of 59	35	45% of 77
Alive with minimal deficit at discharge	33	56% of 59	19	25% of 17
3. Operated — Patients “optimum”	45		34	
Dead	7	16% of 45	6	18% of 34
Alive with minimal deficit at discharge	30	67% of 45	18	53% of 34
4. Operated — Patients “poor-terrible”	7		17	
Dead	5	71% of 7	11	65% of 17
Alive with minimal deficit at discharge	0		0	
5. Carotid ligation alone	38		7	
Dead	6	16% of 38	6	86% of 7
Alive with minimal deficit at discharge	25	66% of 38	1	14% of 7
6. Craniotomy alone	16		69	
Dead	4	25% of 16	29	42% of 69
Alive with minimal deficit at discharge	7	41% of 16	18	26% of 69
7. Carotid ligation and craniotomy	5		1*	
Dead	3	60% of 5		
Alive with minimal deficit at discharge	1	20% of 5		
8. Overall Mortality	21	30% of 71	73	61% of 120
Overall “good result”	42	59% of 71	38	32% of 120

\*Neurologically impaired.

ment of consciousness. Even among those who had evidence of subarachnoid hemorrhage, half were awake and alert on initial examination. This contrasts with a much smaller percentage of similar patients with aneurysms in other locations.

(d) Symptoms and signs were related to the third, fourth, fifth and sixth cranial nerves in a large number of patients in the internal carotid group: i.e. ophthalmoplegia, dilated pupil, ptosis, retro-orbital or facial pain, sensory loss on the face.

These symptoms and signs appear to be due to enlargement of the aneurysm situated adjacent to these cranial nerves in the middle fossa. Most of the aneurysms producing cranial nerve signs were “pointing” medially and posteriorly. Those directed laterally usually did not produce cranial nerve signs. Sudden enlarge-

ment of an aneurysm may suggest dissection and bleeding into its wall without breaking through the adventitia. Occasionally, one will see a large aneurysm in which there is evidence of organizing clot in addition to fresh rupture.

The tendency for internal carotid aneurysms to enlarge was emphasized by Björkstén and Troupp.<sup>3</sup> They pointed out that these aneurysms hang free in a subarachnoid cistern and have a better opportunity for growth than aneurysms supported by enveloping cerebral substance. Out of a series of 700 patients with aneurysms, they reported 19 who had been subjected to a second arteriogram at a later date without intervening surgery. In 10 of these patients the aneurysm had definitely enlarged between examinations, and the most “spectacular” growth was associated with rehemorrhage.

With regard to the 9 patients in the present series with aneurysms in other locations who had "ocular signs" at the time of admission, most of them had a dilated pupil associated with a contralateral hemiplegia and marked impairment of consciousness. These signs were suggestive of third nerve paresis secondary to transtentorial cerebral herniation rather than to local compression by the aneurysm. A number of patients with eyes conjugately deviated to one side, without specific extraocular paresis, were not included under "ocular signs," as this was regarded as indicative of frontal lobe deficit.

(e) McKissock and Walsh<sup>4</sup> reported a 53% mortality in their series of 108 cases of verified aneurysm treated medically. In another study of 51 cases of intracranial aneurysm which were not operated upon, 21 died of initial or recurrent hemorrhage during the first hospital admission, and 7 more died of recurrent hemorrhage.<sup>1</sup>

In the present series, 11% of patients with internal carotid aneurysms and 32% with aneurysms in other locations died without surgery from the initial or recurrent hemorrhage.

(f) Aneurysm surgery is largely prophylactic rather than therapeutic, and once an aneurysm has announced its presence, investigation and treatment should be promptly undertaken in an effort to prevent disastrous rehemorrhage. Proximal carotid ligation in the neck is the treatment of choice in cases of internal carotid aneurysm<sup>1,5</sup> provided the patient has sufficient collateral circulation. This procedure is usually done under local anesthesia with continuous testing of the patient during temporary carotid occlusion. A variety of gradually occluding and releasing arterial clamps have been devised, and current emphasis is upon differential ligation of various branches of the common and external carotid arteries.<sup>6</sup> The hydrodynamic principles underlying this procedure were described by Black and German<sup>7</sup> in 1953, and stasis within an aneurysm following partial carotid occlusion was demonstrated by Ecker and Riemenschneider.<sup>8</sup> Carotid ligation has little place in treatment of other intracranial aneurysms.<sup>7,9</sup>

(g) In the present series of patients with internal carotid aneurysms, all 6 deaths following proximal carotid ligation were due to rehemorrhage within 5 weeks of operation. Two patients with minimal neurologic deficit were subjected to a second arteriogram 6 and 9 years after carotid ligation. Both studies showed complete recanalization of the vessel with obliteration of the aneurysm.

(h) Despite an occasional report of aneurysm rupture during arterial injection<sup>10,11</sup> one should not hesitate to carry out carotid arteriography at any stage in patients with suspected intracranial aneurysm.<sup>5</sup> In a series of 2,332 arteriograms, Feild, et al<sup>10</sup> reported a complication rate of only 2.10%. Most of these complications were mild and transient.

(i) The present study bears out previous observa-

tions by Pool,<sup>12</sup> Poppen and Fager,<sup>1</sup> Hunt, et al<sup>13</sup> that patients in optimum condition at time of surgery fare far better than patients with substantial neurologic impairment.

(j) This study is subject to all the errors inherent in any retrospective study in which the observations of many others must be interpreted after a lapse of time. Some subgroups included in the tables are too small to be of statistical significance, but both the numbers and percentages are given so that the reader is free to draw his own conclusions.

#### SUMMARY

The purpose of this paper has been to emphasize certain clinical aspects of intracranial aneurysms with particular reference to aneurysms of the internal carotid artery. Increasing numbers of these lesions are being detected prior to actual rupture by virtue of recognition of particular signs and symptoms that herald their presence. Seventy-one patients with internal carotid aneurysms are compared with 120 patients with intracranial aneurysms in other locations. Results of surgical procedures are briefly indicated.

#### ACKNOWLEDGMENT

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# Treatable Factors In Chronic Illness

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A year ago, members of the staff of the Thayer Hospital reported a pilot study of patients in nursing homes. In this paper, we will report observations on a different group of patients. These are patients with chronic disease, disability or problems of aging who have prolonged hospital stay or who have recurrent hospital admissions. About 50% of these patients have a course dictated by the severity of their disease, and the amount of hospital utilization cannot be avoided. In the other 50%, disease alone does not account for the hospital utilization. In this group, careful evaluation can often reduce the length of hospital stay and prevent unnecessary readmission. This fact is well documented in the literature.<sup>1-2-3</sup>

In any patient with chronic disease, the diagnosis of factors involved in adapting to the disease is as important as the diagnosis of the disease itself. The ability to cope with disease is an individual matter and is determined by constitutional factors such as age, susceptibility and metabolic state; emotional factors such as motivation and level of anxiety; and environmental factors which are principally connected with the stability of the home. These factors are often amenable to treatment where the primary diagnosis is not. Their treatment may make the difference between unnecessary hospital admission and stabilization of care in the home.

This type of treatment was discussed by Austin Flint as long ago as 1873, in his "Essay on Conservative Medicine." He pointed out that it is important to "conserve the vital forces, to aid the patient in preventing the advance of the ravages of chronic illness and to rehabilitate those who suffer from chronic disability." We have found that the physician will need facilities and services extending from the hospital into the community in order to practice conservative medicine. From our observations of patients, we have developed some of the necessary services and they are briefly summarized in this paper.

## COMMUNITY NURSING SERVICE

The most frequent need for the physician practicing conservative medicine is bedside nursing service in the home. The majority of patients with unnecessarily prolonged hospital stay do not have need for intensive medical care but are kept in the hospital because of their nursing needs. Often these can be carried out in the home by regular visits of the community nurse and education of the family.

In Waterville, there was no public health nurse, or visiting nursing service available on a community basis. In 1960, this need was discussed at a meeting with the Mayor and with the State Commissioner of Health and Welfare. It was decided that the first step towards developing such a service was to appoint a Community Health and Welfare Committee to survey the over-all health needs and to make recommendations to the city government. This committee was duly appointed and consisted of three medical physicians, an osteopathic physician, two councilmen, two aldermen, and two laymen. On its recommendation, the city government voted salary for one community nurse, whose initial duties were bedside care of indigent patients discharged from any of the three hospitals in the community. She has already reached her maximum work-load, and another nurse will be added in the coming year.

The point to be made here is not a justification of community nursing service. This has been documented many times before and, indeed, many other Maine communities have more adequate nursing service than Waterville has as yet. Rather, we wish to emphasize that physicians identified this gap in service, the hospital took the leadership in stimulating the community to act, and physicians took part in planning and carrying through the development of this new service.

## PHYSICAL THERAPY IN THE HOME

Patients with arthritis, Parkinsonism, a fractured hip or a stroke need long-term physical therapy. Once this is started in the hospital, it can be carried on in the home if the family is willing and capable. The Thayer Hospital considers that home care is one of the accepted functions of its physical therapy department.

In any patient with a handicap following a disease or accident, there are residual assets that can be treated even though the handicap cannot be cured. This extra-disease factor is too often overlooked and is the cause of unnecessary loss of function.

The physical therapist on home care finds that it is most effective to focus on education of the family rather than limiting his attention to the patient alone. This assures continuity of care on a day-to-day basis without daily visits, and also has the important function of giving the family a sense of responsibility for the patient's care.

In communities where there is no registered physical therapist, a trained physical therapy aide may be a very successful substitute. In the last two years, the Thayer Hospital has trained several high school graduates as physical therapy aides. Working under the supervision

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of the physician, they can do a good job of setting up continuing activity programs in the home.

#### HOMEMAKERS

One by-product of chronic disease in the elderly is exhaustion. An elderly man was admitted to the Thayer Hospital in a semi-coma. The sole cause was exhaustion resulting from the twenty-four hour care he gave his bedridden wife.

In a young family, a mother's episode of illness may cause the father to lose time at work in order to keep the family together, or may necessitate placement of children in foster homes. These by-products of illness can be avoided by having an established community homemaker service.

The Thayer Hospital — aided by a grant from the State Department of Health and Welfare — has trained two classes of homemakers. Although the hospital saw the need for this service, and was willing to give training courses, it could not finance this community service on a permanent basis. The on-going program in Waterville has been taken over by the Maine Children's Home for Little Wanderers. Reports in the literature show that most community homemaker services are administered by voluntary or public health agencies, and early planning should take this into consideration.

#### RETIRED PERSONS CLUB

For many patients with chronic illness or problems of aging, boredom and lack of social contact can lead to disuse atrophy or to mental confusion. Boredom is an extra-disease factor that can be treated. In Waterville, the problem was discussed with the director of the YMCA. Working with a small group of elderly citizens, he organized a Retired Persons Club. This group meets regularly once a week, and has now grown to 150 members. There is no way of showing statistically what benefit it has had in combatting disuse atrophy and mental confusion. We can only say that it must be answering a need, or the membership would not have increased so rapidly.

#### TRANSPORTATION

There are patients who cannot get to such community facilities as the Retired Persons Club. There are other patients who can continue on home care only if they can get to the hospital for laboratory tests, or medical procedures at regular intervals. The Red Cross has solved this problem by establishing a Motor Corps. In its two years of operation it has proved its effectiveness. A case in point is a man of thirty-two with advanced Parkinsonism. In the past, he had recurrent hospital admissions because he would become unable to walk or even turn over in bed by himself. A few days following admission, he would regain his ability to take full care of himself, to walk unassisted, and even to climb steps. Although the disease was not curable, the extra-disease factors

were reversible. Home physical therapy was not successful because of the patient's superficial motivation and because his wife had to work and leave him alone at home. In the last year, he has not been readmitted to the hospital because the Red Cross Motor Corps has brought him for weekly supervision in the group therapy sessions at the hospital.

#### PHYSICIANS AND COMMUNITY PLANNING

Physicians must be the leaders in planning these community facilities. They are the ones who most clearly see the need for community services. They are the ones who will be referring patients to community services, and without their referrals, no community service can function effectively.

When the physician starts to plan, he will find that he has many sources of help. The Maine Medical Association has an active committee for long-term patient care under the chairmanship of Dr. Peter W. Bowman. This committee is now working with two communities and is eager to work with others. In addition, the State Department of Health and Welfare has a particular interest in this field and some available funds for financial help. Finally, the Bingham Associates Fund through its field representative, Mr. George T. Nilson, can offer valuable assistance. All of these sources of help have been used in developing the program in Waterville.

#### DISCUSSION

The Thayer Hospital staff has been evaluating that group of patients that are prone to prolonged hospital stay or to recurrent hospital admissions. In many, it is not the disease itself, but powerful extra-disease factors that lead to over-utilization of the hospital. These extra-disease factors are amenable to treatment even though the disease itself is not.

In this paper, we have described the most important community facilities that have been developed. They have been chosen for discussion because they are easily reproducible in other communities. These services include:

Home Nursing  
Home Physical Therapy  
Homemaker Services  
Retired Persons Club  
Transportation

Leadership in planning community facilities must come from physicians and the community hospital. When the physician takes this leadership, he will find sources of support are available from several state organizations.

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# Mass Hysteria Masquerading As Food Poisoning

PAUL H. PFEIFFER, M.D.

It was warm for football the afternoon of Saturday, the 19th of October, 1963. Unofficially, the temperature climbed to 84 degrees in the shade. Brewer High School, heavily favored to win, met the home team, Winslow High on the gridiron across the Kennebec from Waterville before an enthusiastic crowd of rooters and both school bands. In a fiercely fought game, Winslow won—and already had a comfortable lead by half time.

The Brewer High School band, wearing heavy woolen uniforms had traveled 56 miles by bus and had eaten lunch at a famous Waterville restaurant. One of the young ladies in the band has a past history of "fainting." Another had been ill the day before, and had felt nauseated during the ride. She spent part of the game in the bus retching. At half time the "faint" girl showed signs of collapsing and was advised, by the Winslow team physician, to return home.

She did not go home at that time.

Of interest is the fact that newspaper reports of an outbreak of Botulinus in Kentucky and New York had attracted much attention the preceding week. This may have influenced the events which followed.

I was finishing rounds at Thayer Hospital about 3:00 p.m., when a frantic voice from the accident room informed me that 8 high school students were being rushed over by ambulance with "food poisoning." Before I had a chance to hang up and ponder this news, the siren of the police car announced the arrival of the victims. Bedlam ensued. Nurses and aides soon had 8 girls on stretchers in and about the emergency room. The girls screamed about numb hands and abdominal pains caused them to writhe violently; but only one (who had previously been ill as mentioned) showed signs of vomiting. Excessive ventilation with prompt development of the carpopedal spasms noted in alkalotic tetany developed. Sweating, tachycardia, hypotension and shock were absent.

To the consternation of the older and wiser nurses,

emetics and stomach pumps were withheld until a clearer clinical picture was demonstrated.

In the meantime, rebreathing of CO<sub>2</sub> in paper and plastic bags seemed to help some of the girls regain their senses. Two recovered rapidly and described some of the details mentioned above.

The triggering incident was never discovered. Had some suggestible individual noted the fainting and the retching girls and hastily drawn the conclusion that this must be food poisoning? Were they anquished by the stunning defeat being suffered by their favorites; and needed distraction to obscure this painful actuality?

Noting that the four girls who were inside the emergency room seemed to feed each others' hysteria, an alert supervisor moved the girls into the wards where they could be separated. The underlying fear of contaminated food ingestion remained strong enough that none were put on the maternity ward, however.

Over the course of an hour or two and with the help of rebreathing and a barbiturate injection the girls regained their equilibrium and calmly awaited the arrival of their parents.

It was remarkable at this point to observe that several of the girls were stable and sensible people — correlating poorly with their shenanigans of a few moments earlier.

What kept the reaction from spreading to the other two or three hundred young ladies? Several of the victims were horn players although one of the sickest played the cymbals. Was this a physiological response to the discomfort of a thick, woolen, unbecoming suit on a hot day combined with overbreathing from cheering and blowing into trumpets or was it purely psychological mass hysteria?

My knowledge of the mechanisms responsible for mass hysteria is insufficient to answer this question.

# The End Of The Waiting List In Mental Retardation?

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There have been few problems in State Government during the past 25 years that can match the persistent political annoyances and irritations, the professional frustrations, and the parental desperation resulting from the long, almost stagnant waiting lists of the hospitals for children and adults with mental retardation.

The words "waiting list" have become an enigma to all concerned. They have been used to "explain" the lack of funds, and the denial of essential diagnostic and therapeutic services in our institutions. They have been used to present astronomical demands for new construction and operating budgets to legislators. And, worst of all, they are a natural alibi to explain (and justify) inaction and inertia that have prevailed so long in too many places.

It is our contention that the existing waiting lists, preventing children, often for periods of one or two years, from receiving diagnostic and therapeutic services, are unnecessary and can be eliminated by means other than spending many millions for new hospital bed space and many more millions for cumbersome and expensive operations thereby creating yet another stagnant problem for state governments.

The waiting list is the product of scientific advances leading to a very low infant, child, and adult, mortality rate in this country, thereby resulting in a significantly higher survival rate, not only for all groups of patients, but particularly among children with the mental retardation syndrome. Their number has increased and is increasing unproportionately when compared with death rates 30 years ago.

This development culminated when the concepts of segregation and lifelong custodial care were the stereotyped answers that eventually led to facilities virtually that of the size of towns and assuming some of the characteristics of ghettos.

The parental and professional revolution of the 1950's that accompanied the availability of new knowledge, skills, and facilities, has brought an end to this infamous era. Where it still survives it will continue to display symptoms of fiscal, professional, and emotional incompatibility until replaced by more rational and valid solutions.

It is somewhat surprising to realize that the development of more appropriate approaches to mental retardation than the waiting list has been slow.

For many years, for example, diagnostic out-patient clinic services<sup>1</sup> have been in operation that have offered specific recommendations for the management of the child with mental retardation in the community.

A limiting factor in previous years was, of course, the fact that community resources were non-existent in most places so that placement on the waiting list was still the inevitable fate of most children in those days.

Today, however, this has changed and the pioneering work of the Fernald Group has gained new significance. It is already reflected in the establishment of diagnostic clinics for retarded pre-school and school children in many places.<sup>2</sup>

In midsummer of 1963, we decided to make a concentrated effort to see objectively who was on the waiting list and why.<sup>3</sup> Based upon the experience in our weekly diagnostic staff conference over many years, we knew that there were cases on the waiting list that did not require residential care. We knew of cases on the waiting list that were social emergencies, where good community casework was indicated, not institutionalization. And, we knew that a number of cases, particularly in the dependent group, required primarily good nursing care and could not possibly utilize professional services available at our hospital.

It should be stated that we are reporting from a State with a stable population (approximately 1,000,000). During the past 5 years we have consistently discharged more patients than we have admitted, decreasing thereby our daily average census from an all time high of 1510, several years ago, to a daily average of 1194 in November of 1963. It is anticipated that this trend will continue although we do not feel qualified to make firm predictions.

It should also be stated that the waiting list in Maine has consistently fluctuated from 100 to 200 applicants at any given time since 1953.

The method of our approach was admission of all cases from the waiting list at a rate of from three to five patients per week for 35 days of observation. We started with the educable applicants on the assumption that some, if not many, would be logical candidates for special classes in the community and that they should be enrolled there, with our recommendations, when school would start shortly after Labor Day.

\*Superintendent and Director of Research, Pineland Hospital and Training Center, Pownal, Maine.

\*\*Director of Para-Medical Services, Pineland Hospital and Training Center, Pownal, Maine.



As of January 1, 1964, 46 patients had been admitted for 35 days, studied, evaluated, and staffed, under this new approach so that constructive rehabilitation disposition could be effected.

The breakdown of this caseload is quite interesting and certainly there is a trend in concurrence with the findings of the Onondaga Study,<sup>4</sup> in regard to mental retardation and its relationship to chronological age. It appears that the way in which our society evaluates a youngster for special class attendance also holds true for residential school admittance, namely, that retardation is a function of either the inability to be academically proficient or the inability to be economically sufficient or valuable.

The chronological age range of admissions was from 5 years 3 months to 39 years 2 months (Pineland presently does not admit patients under the age of 5 due to lack of a pediatric unit for infants and pre-school children, which had been, and will be again, requested).

Table I reflects the age category of the 46 admissions to Pineland since this new approach to the waiting list began last summer.

TABLE I

<i>Chronological Age</i>	<i>Number of Patients Admitted</i>
5- 9	12
10-14	16
15-17	9
18-20	5
20-22	1
Over	3

It would appear that Table I tends to point out that there is a lack of valid identification tools or more of a passive attitude toward behavior up to the age of 10 on the part of teachers and parents. However, between the ages of 10 and 14, it appears that both parents' and teachers' frustration levels reach their peak.

After this critical period, when a youngster can and does show a little usefulness, or, as happens in most cases, when he is legally excluded from school, which, incidentally, removes him from a frustrating or conflict situation, the number of cases referred for admission begins to drop. It is interesting to note that only 1 case referred for admission was between the ages of 20 and 32, inclusive, and only 3 from 32 to 40.

When we reach this last group of referred patients, we find that, in general, these older patients are referred either for fear of committing, or actual commission of, anti-social acts. Also prevalent may be the parental anxiety over "what is to become of him" after they will have passed on in view of the fact that their child may not be economically self-sufficient or socially adaptable without supervision.

The intelligence quotient of the group ranged from an SMQ of 5 to a Full Scale Weschler IQ of 92 and

with a Mean of 51.1. The patient with the 92 IQ was retarded, not in mental ability but in physical ability brought about by progressive myositis ossificans, and is, therefore, a candidate for physical medicine rehabilitation and regular education rather than for residential rehabilitation.

Table II indicates a breakdown in the intellectual ability of the 46 admitted patients as tested by staff psychologists:

TABLE II

<i>IQ Range</i>	<i>Number of Patients</i>
90-99	1
80-89	1
70-79	3
60-69	10
50-59	11
40-49	9
30-39	4
20-29	5
10-19	0
0- 9	2

Table III breaks down the 46 patients into percentages and educational classifications:

TABLE III

<i>Educational Classification</i>	<i>Percentage</i>
Dependent	4.3%
Trainable	39.1%
Educable	45.7%
Slow Learner	8.7%
Low Normal	2.2%

Of the total group, all but 15.2% should be educationally provided for in the community if the permissive legislation on educables and the Attorney General's ruling on subsidy for community trainable classes could be fully taken advantage of within the State of Maine.

If one leaves the trainable group out, still 56.6%, or all those whose intellectual capacity is in the educable or intellectually higher range, are definitely the responsibility of the community and should not have to come under the hospital's treatment program. This, of course, does not infer that all 56.6% can, at this point, be included in community special classes, for, unfortunately, Maine has not arrived at that level of educational development. For that reason we tended to be realistic with our recommendations so that 20 of the 46 patients (of whom 10 are educables and admitted under this new approach) are being kept on at Pineland Hospital for further training because the community from which the child comes does not as yet

have the necessary educational facilities. This amounts to 50% of the educables who have been seen up to this point.

The remaining 10, who are being kept at the hospital, are being retained for a number of reasons, some of which are as follows:

Further observation, so that more realistic appraisals and recommendations may be made; 6 months training program so that behavior might be controlled to the point where the youngster might become better tolerated at home; chemotherapy to modify behavioral adjustment; counseling of patients as well as of parents, so that more integrated and realistic plans may be effected; planning for inclusion in the State Vocational Rehabilitation program with Woodrow Wilson Rehabilitation Center.

Along with the above, those youngsters who remained at Pineland had one, or a combination, of the following types of programs assigned to them, depending upon their needs:

Special services such as the teaching of tasks of daily living for low trainables and dependents, if possible;

speech therapy, occupational therapy, physical therapy, vocational education, school, and driver education, for the rest.

Recommendations for the 26 who were returned to the community included:

Change of domicile for 2 patients from their own homes to foster home placement (this was done for the benefit of the patients involved); 3 patients were recommended to make application for Aid to the Permanently and Totally Disabled, as well as 1 for nursing home care. In the case of the individual with the 92 IQ, the recommendation included a work-up in the area of rehabilitative physical medicine as well as the actual therapeutic course to be followed. Parent Counseling was recommended in 12 cases so that more acceptability might be engendered in the home. Anti-convulsant medication was recommended for 3 patients, while tranquilizers were recommended for 2 of the returnees.

Table IV indicates most of the recommendations made in line with what the respective communities *now* have to offer:

TABLE IV

<i>Special Classes</i>	<i>Vocational Rehab.</i>	<i>Sheltered Workshop</i>	<i>Speech Therapy</i>	<i>Remedial Reading</i>	<i>Parent Counsel.</i>	<i>Patient Counsel.</i>	<i>Medica- tion</i>
14 (8 educ. 5 train. 1 regular)	3	6	2	1	12	3	5

Undoubtedly, it is too early yet to make any firm prediction as to the validity of the above trend; however, it is certain that the following needs of the parents have been met:

- (1) Somebody does care about the difficulty that they are having, and is willing to do something about it.
- (2) Closure in the area of planning and future goals for their youngsters.
- (3) An unspecified realization by at least two sets of parents that the child does meet a need of theirs in just being in the home.
- (4) They have received professional referrals to existing agencies in the community which they might or might not have known about but which, in any case, would thereby appear to have attained greater effectiveness.

This program also contributes through the Pineland Staff recommendations a heightening of interest in developing needed community resources.

Another 42 patients have been accepted for admission and the remaining applicants now on the waiting list will have been evaluated by midsummer of this year.

From then on, we shall be able to process future applicants within a few weeks after initial admission has

been sought since the average number of new applications has been 11.5 per month for the last 7 years.

This gratifying accomplishment has been made possible by the availability of a qualified Staff\* of experienced specialists who accepted, willingly, this additional challenge and by the administrative and legislative leadership in Maine, and nationally, that laid the foundations for essential community participation and development of community resources during the past 8 years. (Special classes, Vocational Rehabilitation, Aid to the Totally and Permanently Disabled).

This highly significant development tempts us to project some important conclusions into future planning in the field of mental retardation. We can terminate expansion at Pineland Hospital of dormitory space in favor of regional nursing homes and modified boarding homes that will serve chronically ill patients whose disabilities, however, do not require hospital services.

We can, therefore, plan for operating budgets to staff and maintain these nursing and boarding homes.

\*We must give special credit to Dr. Earle B. Perkins and Mr. John J. O'Toole, ACSW as well as the members of the Department of Psychiatric Social Service, whose skillful and persistent labors contributed greatly to the success.





DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### Syphilis Still Challenges Medicine\*

WILLIAM J. BROWN, M.D.\*\*

In 1947, more than 106,500 cases of early infectious syphilis were reported in the United States. Eight years later, this figure had been reduced by 100,000 to a low of 6,500 — surely one of the greatest medical advances of all time. Penicillin was hailed as the doom speller of syphilis and gonorrhea, and it was agreed among most authorities that the eradication of both diseases was imminent.

By 1953, almost all in-patient treatment for venereal disease was terminated; and Federal venereal disease control appropriations, which had once topped \$17,000,000 were reduced to a low of \$3,000,000 in 1955. That same year, the late esteemed Paul O'Leary dropped 'and Syphilology' from the A.M.A. Archives of Dermatology and Syphilology.

Syphilology was de-emphasized in the curricula of medical schools throughout the country; and thousands of young doctors were graduated without ever having probed the depths of meaning behind Sir William Osler's famous predication that, "To know syphilis is to know medicine."

The Board of Commissioners of the Joint Commission on Accreditation of Hospitals dropped routine admission serologic tests for syphilis as a requirement for accreditation on January 28, 1956, and many authorities proclaimed the premarital blood test requirement a useless relic.

In fact, penicillin ushered in the era of the wonder drug with such beguiling promise that medic and layman alike could hardly be blamed for having been lulled into apathetic optimism that can only now be called, through benefit of hindsight, premature.

And premature it was, for in 1956 the treponeme began to reassert itself; 6,251 cases of infectious syphilis being reported in 1957; 6,661 in 1958; 8,178 in 1959; and so on, until in 1962, more than 20,000 cases were reported. And this represents only a fraction of the cases which were actually diagnosed, let alone of total incidence.

Gradually, the resurgence of syphilis having finally

impressed itself upon the forces of public health as a trend rather than a vagary, these forces dramatically re-girded for battle. New control techniques were developed to meet new problems, and through increased control appropriations, badly depleted staffs were supplemented with new people trained in the newest techniques. But a generation of physicians had gone into private practice, most of whom had never seen a syphilitic lesion. And more were, and are, being graduated annually who have never been exposed to more than a scant few hours of syphilology.

What are some of the results?

One result is reflected in the number of reports of patients exhibiting classic signs of syphilis but diagnosed as suffering from an allergy or from some other benign dermatosis and treated accordingly, without consideration of the possibility of syphilis, principally because the patient was well dressed and educated and 'above suspicion of syphilis.' The tragedy is that in most such cases syphilis was not deliberately excluded from differential diagnosis; it was just not even considered.

Moreover, given a similar patient and a reactive serologic test for syphilis, there is often a tendency to interpret the serology as a biologic false positive reaction for the same reason. In such cases, even if 'insurance' therapy is administered, the patient usually is lost to epidemiology.

But to go a step further, repeated studies have demonstrated that for every privately treated case reported, many have been treated and not reported because, among other things, the possible embarrassment of the patient is adjudged more important than the lives of the contacts or the health of the community.

Yet every case of infectious syphilis, like every case of typhus, typhoid, polio, or plague, is important for epidemiology if the disease is to be eradicated.

While many private physicians do ask the patient about sexual contacts, most cannot spare the extra hour or more of time required for a thorough probe for contacts, which often discloses a surprising number. And the search for these contacts, which may extend over several states and countries, definitely is beyond the scope of the busy physician's activity.

The eradication of syphilis is a joint responsibility; and we in public health look forward to the day when

*Continued on Page 36*

\* Reprinted from Journal of Chronic Diseases, 1963, Vol. 16 pp 1043-1045 through the courtesy of the Co-Editor, Louis Lasagna, M.D. and the author.

\*\* Chief, Venereal Disease Branch, Communicable Disease Center, Public Health Service, U.S. Department of Health, Education and Welfare.

## *Maine Heart Association Notes*—————



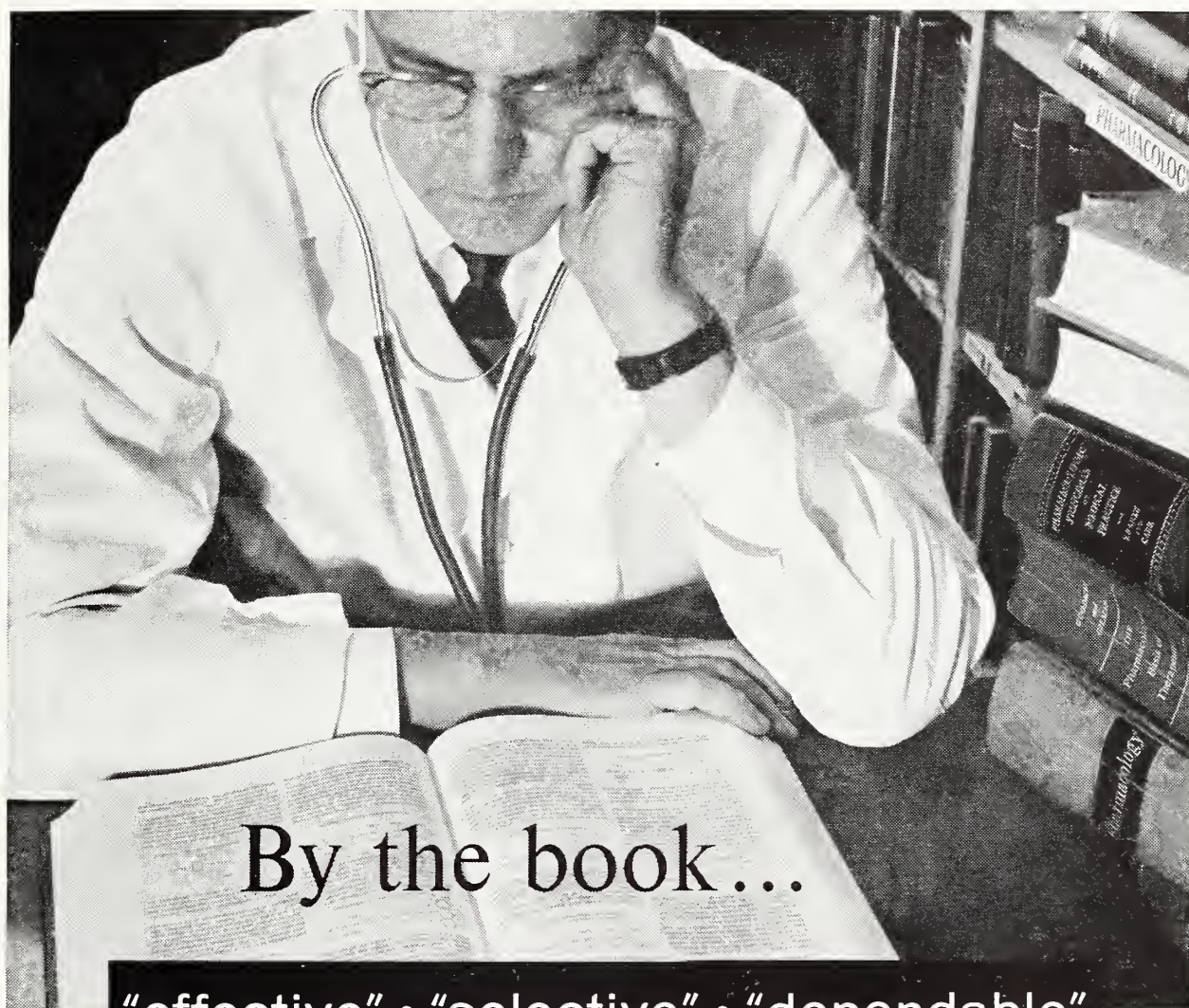
### **Postgraduate Course on Streptococcal Diseases at Bangor**

A series of postgraduate lectures on streptococcal diseases will be held at the Eastern Maine General Hospital, Bangor, in March, according to Dr. Milan A. Chapin, Auburn, chairman of the Maine Heart Association's committee for Professional Education. The lectures, which are co-sponsored by the Eastern Maine General Hospital and the Maine Heart Association, are scheduled for April 1, 8, 15, 22, will be attended by physicians from all parts of the state who are interested in streptococcal diseases with special emphasis on rheumatic fever and chronic valvular heart disease.

The lecturers and their specialized subjects will be Louis Weinstein, M.D., Professor of Medicine, Tufts University School of Medicine, "Streptococcal Infections and Diseases-Rheumatic Fever"; Ralph A. Deterling, M.D., Professor of Surgery, Tufts University School of Medicine, "Rheumatic and Valvular Heart Disease, Surgical Aspects — Cardiac Catheterization"; David Littman, M.D., Chief Cardiology Department U. S. Veterans Administration Hospital, West Roxbury, Mass., will speak at two lecture periods on "Rheumatic Fever and Heart Disease; Congestive Heart Failure; Drugs Commonly Used in Treatment; Arrhythmias" and will close the series with a general discussion on the use of the electrocardiograph.

Dr. Chapin said the course will comprise 12 hours and is designed to be of value to all practicing physicians. Registration information can be obtained by writing Dr. John Bjorn, Coordinator, Eastern Maine General Hospital, Bangor, Me.





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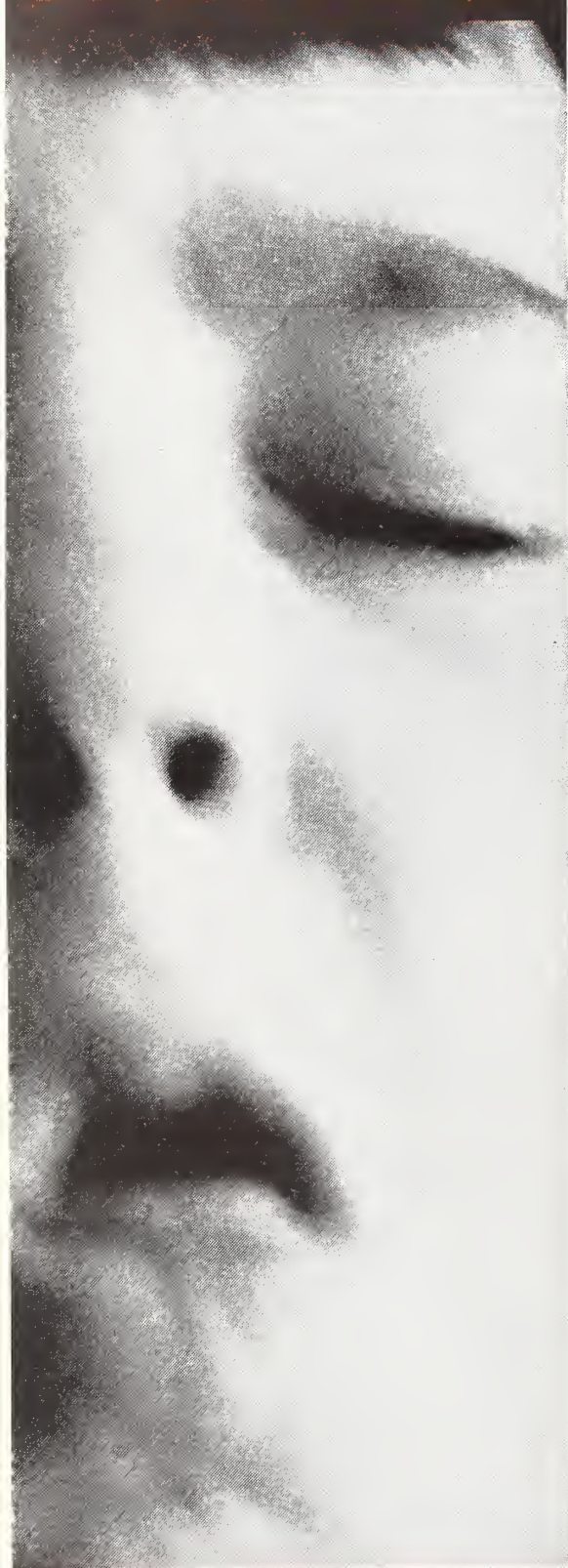
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## News, Notes and Announcements

### Postgraduate-Refresher Course Specific Methods of Treatment

Sponsored by the Medical Staff  
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Sessions will be held in Hiebert Hall at the hospital  
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March 4, 1964—*Diagnosis and Treatment of Auto-immune Hemolytic Anemia*

WILLIAM DAMESHEK, M.D.

Professor of Medicine, Tufts University School of Medicine; Chief of Medical Services, Lemuel Shattuck Hospital, Boston; Department of Public Health, Commonwealth of Massachusetts, Boston, Massachusetts.

March 11, 1964—*Diagnosis and Treatment of Addison's Disease*

DAVID P. LAULER, M.D.

Assistant in Medicine, Peter Bent Brigham Hospital, Boston; Research Associate in Medicine, Harvard Medical School.

March 18, 1964—*Diagnosis and Treatment of Hyper- and Hypothyroidism*

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Assistant Physician, Pratt Clinic-New England Center Hospital, Boston; Senior Instructor in Medicine, Tufts University School of Medicine.

March 25, 1964—*Modern Treatment of Coronary Occlusion and Insufficiency*

WILLIAM C. ELLIOTT, M.D.

Assistant in Medicine, Peter Bent Brigham Hospital; Research Fellow in Medicine, Harvard Medical School.

April 1, 1964—*Diagnosis and Treatment of Venous Thrombosis and Pulmonary Embolisms*

DUNCAN P. THOMAS, M.D., Ph.D.

Associate in Medicine, Beth Israel Hospital, Boston; Instructor in Medicine, Harvard Medical School.



April 8, 1964—*The Treatment of Diabetes Mellitus*  
PRISCILLA WHITE, M.D., F.A.C.P.  
Joslin Clinic, Boston; Assistant Professor of Pediatrics,  
Tufts University School of Medicine.  
Registration Fee: \$30.00. Application has been made to the  
American Academy of General Practice for postgraduate credit  
under Category I.

The End Of The Waiting List In Mental Retardation?  
*Continued from Page 30*

This can be done on a private or tax-supported basis and will be far less expensive than new hospital staff and facilities.

We can thus free our hospital staff to the point where more and more productive and effective diagnostic and therapeutic services will be rendered and thereby give more and better services to those patients who need them and at a time when they need them.

REFERENCES

1. The Walter E. Fernald State School, Waltham, Massachusetts, has operated such a clinic for several decades.
2. Haskell, Eliz. N. et al. The First Three Years of a Clinic for Mentally Retarded Pre-School Children. J.M.M.A. 1961 52:47.
3. We wish to express our thanks to Commissioner Walter F. Ulmer, Dr. Wm. E. Schumacher, Director of Mental Health, and to Dr. Earle B. Perkins, Director of Admissions, all of the Department of Mental Health and Corrections, for their support and assistance.
4. (Onondaga County Survey (1955) 'A special census of suspected-referred mental retardation.' Community ment. Hlth.Res., New York State Dept. Ment. Hyg. Rep.)

Internal Carotid Aneurysms "A Different Breed Of Cat"  
*Continued from Page 24*

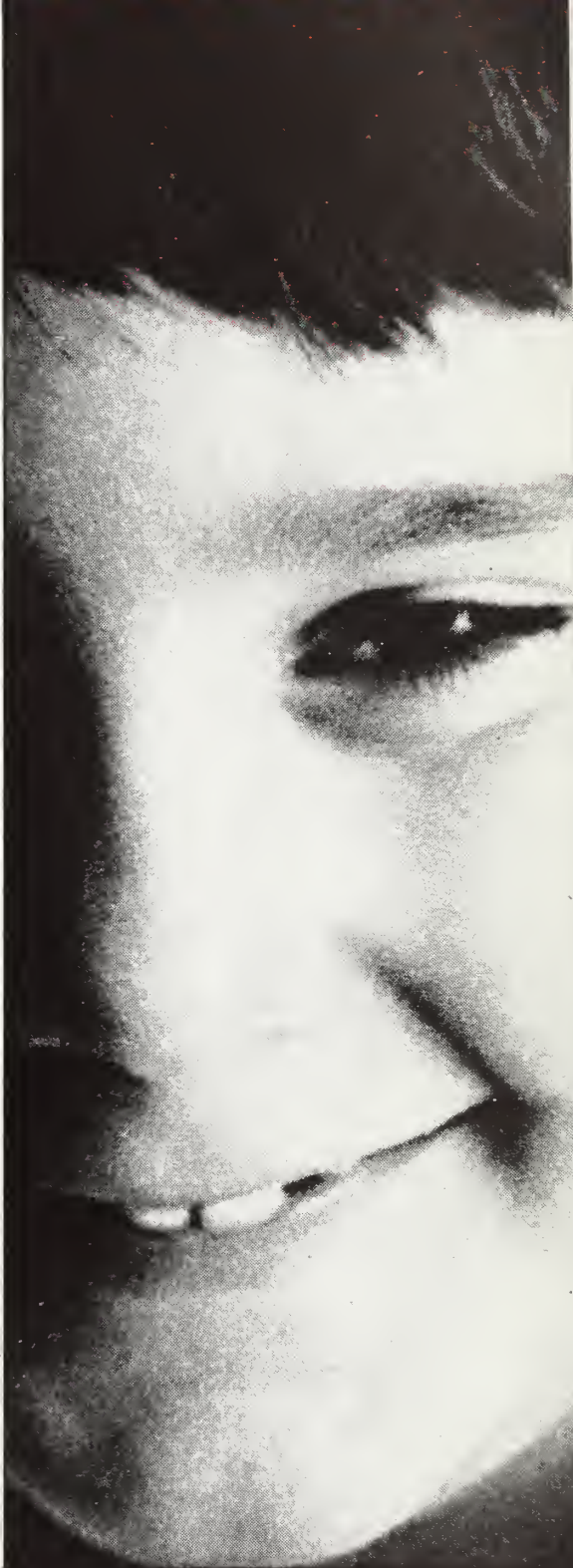
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13. Hunt, W. E., Meagher, J. N., and Barnes, J. E.: The Management of Intracranial Aneurysm. Journal of Neurosurgery, 19: 34-40, 1962.

33 College Avenue, Waterville, Maine

Correction

Clinico-Pathological Conference. — In the CPC published in the Dec. 1963 issue (Vol. 54, page 257), the following statement made by Dr. Robert O. Kellogg "Gross hematuria was present, but subsided on steroids, as did her bloody diarrhea," should have been printed at the end of the CPC as Dr. Kellogg was not present at the time the CPC was given but offered the information at a later date.

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DEPARTMENT OF HEALTH AND WELFARE — *Continued from Page 31*

there is a clear-cut partnership between private medicine and public health — a partnership in which all patients are diagnosed and treated by private physicians who will then call upon the services of trained public health epidemiologists to interview patients confidentially, trace contacts diligently, approach them carefully, and refer them back to the private physicians for diagnosis, and treatment if necessary.

Without complete morbidity reporting, epidemiologic follow-up, and a sense of urgency and intolerability for the disease, the eradication of syphilis is a difficult goal at best. The Public Health Service is making every effort to provide through State and local health departments highly trained epidemiologic assistance to every doctor in every area of the United States. But this assistance, these skills, must be used universally if they are to be effective against syphilis. And it is good to see the trend moving in this direction.

More and more private physicians are becoming aware of syphilis, and including it in differential diagnosis, are reporting morbidity, and are utilizing the skills available to them through local health department auspices.

But the old interest in syphilology can only come from a return of the subject matter to the medical school curriculum.

Syphilis is a fascinating subject, still presenting many unsolved problems to challenge keen young minds.

Among the infectious diseases, syphilis probably ranks first in the complexity of its manifestations and of its immunological mechanisms. It is replete with features seemingly unique in the repertoire of human infections. Osler's epigram might well be paraphrased: "To understand syphilis is to understand immunology." The gaps in our knowledge of immune mechanisms are shown more clearly in the study of syphilis than any other single disease.

Ten years of widespread belief in the erroneous assumption that penicillin would eradicate syphilis have passed. Syphilis will probably be eradicated through the combined efforts of public health and private medicine within the decade to come. But the job will not be easy. Knowledge will have to replace ignorance; awareness apathy. And eradication will have to be recognized as the only criterion of the success of our efforts.

## New VD Control Service Available For Maine Physicians

The first step in an expanded VD Disease Control program in Maine is starting with a series of personal visits to physicians by William M. Kitching, Field Epidemiologist with the U.S. Public Health Service who is currently assigned to the Maine Department of Health and Welfare. Objectives of the Maine program are:

To acquaint Maine physicians with the new program and with current literature on the general subject of venereal disease;

Familiarize Maine physicians with the facilities available in the State Bureau of Health;

Encourage morbidity reporting;

Seek the cooperation of the private physician in allowing all his early syphilis patients to be discreetly, fully and tactfully interviewed;

Inform physicians of the services available on request for the epidemiology of early syphilis, e.g., if a physician has a patient with lesions suspicious of early syphilis, a dark

field microscope can be delivered to his office upon request. This particular service will be available within the limitations of time and distance.

Mr. Kitching comes to Maine with a background of several years of training and experience in this work. He is a native of South Carolina; received a bachelor's degree in psychology from the University of South Carolina in 1959; served in the U.S. Army Reserve and taught school briefly before joining the South Carolina State Board of Health as a VD investigator. He joined the staff of the VD Branch of the U.S. Public Health Service in February, 1962 and was assigned to the Division of Social Hygiene, New York City Department of Health where he was stationed until his present assignment in Maine. Mr. Kitching's headquarters are in the central office of the Department of Health and Welfare, State House, Augusta and he may be reached by telephone at 623-4511 — Extension 600.

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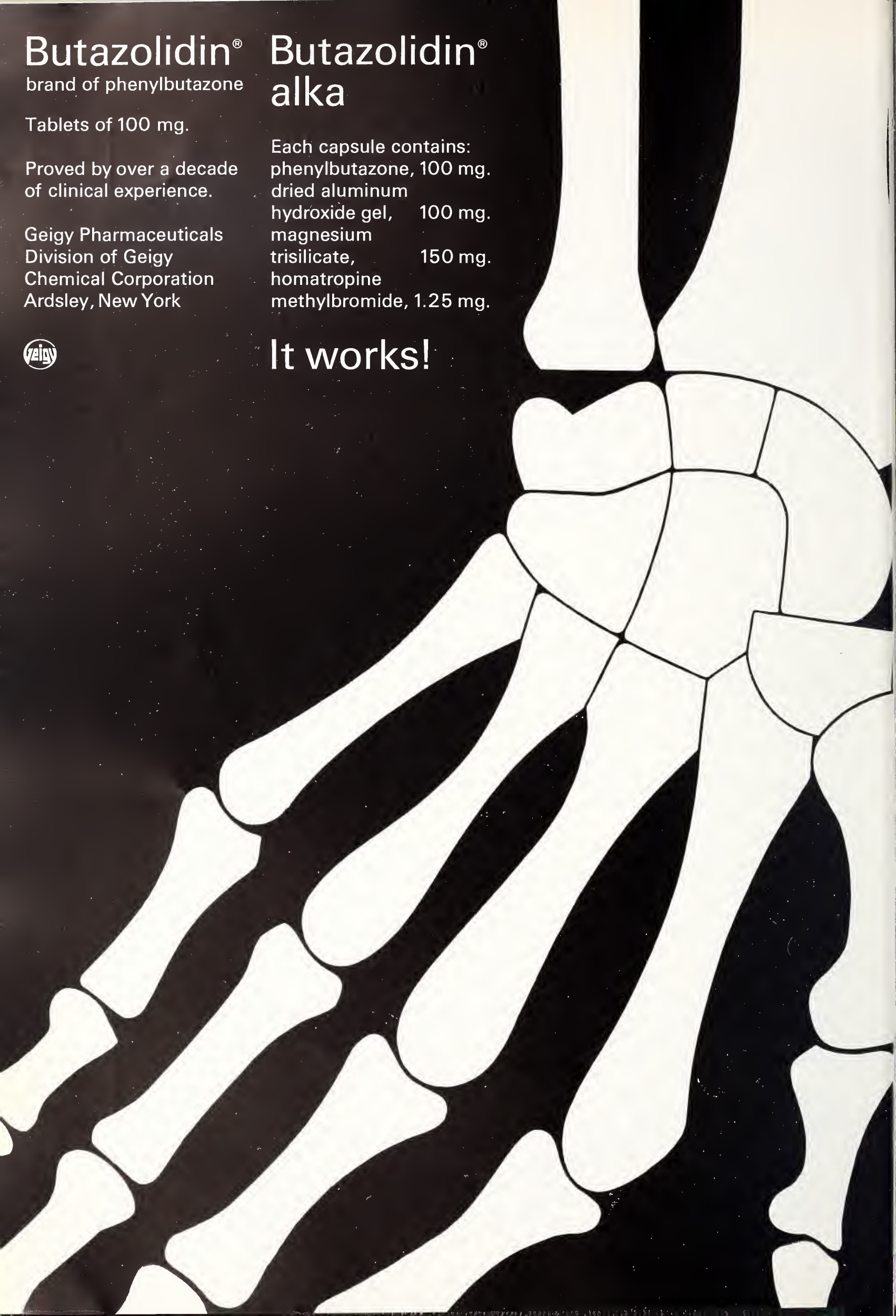
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# The Journal of the Maine Medical Association

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No. 3

## Pigmented Neoplasms Of The Iris\*

RONALD S. POTTS, M.D., C.M.\*\*

There are two reasons which prompt this subject: the obvious easy accessibility of this organ to both casual and expert examination; and the recent conservative surgical trend in approaching these problems. Previously, if involvement of the anterior filtration angle or the ciliary body could be demonstrated, this was automatically considered indication for enucleation. It is now realized that with adequate biopsy diagnosis and proper knowledge of the natural history, many of these neoplasms can simply be closely observed or if indicated can be adequately locally excised by partial iridectomy or partial iridocyclectomy; enucleation now has limited and strictly defined indications.<sup>2</sup>

Much of this change in thinking and management is the result of the technique of gonioscopy. This is a means whereby a contact lens is placed upon the cornea and light rays refracted through it almost at a right angle into the anterior chamber filtration angle. In this way the entire circumference of the angle can be closely scrutinized.

Pigmented iris neoplasms form a spectrum of diseases with freckles and true melanomas merging at one end, and true melanomas then merging with melanocarcinomas at the other. Freckles and melanomas are probably always congenital. Because of small size or slight pigmentation they may not become evident until after puberty or pregnancy. There is also a curious tendency for them to be larger, more numerous, and more pigmented in eyes that already harbor a melanocarcinoma. Freckles and melanomas are very common, found in at

least 50% of otherwise normal eyes.<sup>1</sup> In order of decreasing site frequency, they are found in the iris, ciliary body, and choroid. Freckles obviously are not neoplasms; they are simply local accumulations of melanin-containing cells within the stroma of the iris. They are mentioned here because they must be differentiated from neoplasms. Melanomas need only be properly diagnosed by gonioscopy and surgical biopsy, and then closely followed by periodic examination to first detect any change in size, shape, or pigmentation. If this occurs, they are then properly treated by adequate local surgical excision; enucleation is not necessary.<sup>2</sup>

Melanocarcinomas are the most common intra-ocular malignant neoplasms, although accounting for only about 0.05% of all malignant neoplasms from any body site.<sup>1,3</sup> They are never congenital and almost never bilateral. Most people feel they arise from existing melanomas; hence the urgency for closely following proven melanomas. Melanocarcinoma is very rare in children, the peak incidence occurring by the fifth and sixth decade with progressive increase in incidence through the ninth decade. In increasing order of site frequency, they are found in the iris, ciliary body, and choroid. The most frequent tumors and neoplasms of the iris are innately benign diseases and hence the import of therapeutic conservatism.

Melanocarcinomas assume three histological forms which are of prognostic significance. The most frequent form in the iris fortunately is the spindle cell type. This has a 5 year mortality rate of 5 to 15% and a 10 year mortality rate of 35%. The epithelioid cell type and the mixed spindle cell-epithelioid cell type have 5 year mortality rates of 50 to 60% and 10 year mortality rates of about 82%.<sup>1</sup> Adequate histological diagnosis is thus not simply of academic interest.

\*Presented at the Tumor Clinic, Central Maine General Hospital on November 26, 1963.

\*\* Associate Pathologist, Central Maine General Hospital, Lewiston, Maine.

Melanocarcinomas of the iris tend to assume two clinical patterns. Most commonly, they are well localized and fortunately are usually of spindle cell type. There is a predilection to arise in the lower temporal sector. Arising within the stroma of the iris, they are limited posteriorly by the dilator muscle and the posterior pigment epithelium. They thus tend to grow anteriorly, projecting into the anterior chamber or the pupil. If located near the pupil margin or the midzonal iris region, they may distort the pupil and early impair the patient's vision. If located peripherally, they may extend locally into the anterior filtration angle. If long undetected, due to the characteristically slow but progressive growth, they may then extend circumferentially around the anterior filtration angle to eventually cause secondary glaucoma. The spindle cell melanocarcinoma is very cohesive; the cells are not easily shed or disseminated. If glaucoma is not present at the time of diagnosis this is the form which lends itself most happily to adequate local surgical excision.<sup>2</sup> If glaucoma is already present, then enucleation is the only recourse.

Although the spindle cell melanocarcinoma seldom metastasizes within or without the eye, it is characteristically locally invasive. The growth is slow and relentless, and this is most evident if excision is inadequate. Many years may elapse between the time of surgery and the manifested recurrence, a period during which both patient and physician may be lulled into false security. By the time recurrence is evident, it is usually as a result of secondary glaucoma and here again enucleation is the only recourse.

Very rarely a localized melanocarcinoma consists of the epithelioid or mixed cell types. These cells are not very cohesive and are easily shed into the aqueous humor of the anterior chamber, through which they are disseminated. They tend to settle dependently, choke the anterior filtration angle, and be spread circumferentially. These cell types early result in glaucoma and enucleation is the only recourse: because secondary changes in the eye are usually already severe; and because there is no way to be certain all the cells are removed by local excision.

The second clinical pattern is the diffuse melanocarcinoma. Fortunately this is not very frequent and unfortunately usually is of the mixed or epithelioid cell types. It must be differentiated from a completely benign, nonneoplastic hyperpigmentation called melanosis oculi.<sup>2</sup> This is a congenital uniocular hyperpigmentation

of the iris, ciliary body and choroid, sclera, optic disc, leptomeninges of the optic nerve, conjunctiva, and peri-orbital skin. In typical form it is not easily confused with a diffuse melanocarcinoma. Occasionally, however, melanosis exists only as segmental hyperpigmentation of the iris and this is where confusion may occur. The history is all important in making the distinction. Melanosis is congenital and unchanging. Diffuse melanocarcinoma is so insidious and pernicious that the patient may not even have been aware of increasing pigmentation, and probably will not be able to tell when it first did become evident. Careful slit-lamp examination reveals that melanosis is diffuse, uniform, and does not distort or obliterate normal iris markings. Diffuse melanocarcinoma characteristically produces blotchy irregular hyperpigmentation and, due to iris thickening by the proliferating neoplasm, tends to obliterate iris markings.

Since the diffuse form of melanocarcinoma is most likely the epithelioid or mixed cell types, secondary glaucoma is an early complication. An eye already glaucomatous with hyperpigmentation of the ipsilateral iris should be suspected of harboring a diffuse melanocarcinoma until proven otherwise.<sup>2</sup> This combination of signs is not pathognomonic because it may also result from a ferrous foreign body, or from iris hemosiderosis resulting from an old intra-ocular hemorrhage. The treatment for the diffuse melanocarcinoma is enucleation since the eye is probably already severely damaged and there is no way of reliably stating that all the tumor has been excised.

With this resumé, it can be realized the most common pigmented neoplasms of a readily observed organ are benign. Adequate local excision is all that need be done, and then only if there is manifest change in the recorded disease. Many eyes can thusly be retained and salvaged. Even the usual type of the less frequently occurring melanocarcinoma does not dictate enucleation if diagnosed before the late onset of secondary glaucoma. A few lives may be saved by earlier detection of secondary glaucoma; but more lives and eyes will be saved by proper management of earlier diagnosed iris neoplasms.

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# The Determination Of Serum Total Cholesterol

## Evaluation Of A Simplified Method

MILAN A. CHAPIN, M.D.\*

The serum total cholesterol is a laboratory measurement which is frequently used in clinical medicine. This is particularly so because it is thought to represent the altered lipid state encountered in atherosclerosis and coronary heart disease. An elevated serum cholesterol is recognized as one of the measurable factors, in association with hypertension, obesity, etc., which suggests susceptibility to coronary disease.<sup>1</sup>

Accurate measurement of the serum cholesterol has, in most laboratories, been accomplished by the Schoenheimer-Sperry method.<sup>2</sup> This involves extraction of the cholesterol in both free and ester forms, saponification to convert the esters to free cholesterol, precipitation of the latter with digitonin, adequate washing to remove excess digitonin, and final application of the Liebermann-Burchard reagents for color formation. This method, while accurate, is highly technical, involves many steps and the better part of two days time. It is

done only by a skilled technician. In spite of this, the results may vary in different laboratories.<sup>3</sup> These features, in addition to the elaborate equipment, make this a somewhat expensive procedure and one not usually accomplished in a physician's office. Several other technics for the determination of serum cholesterol have appeared in the literature and Table I lists several of these.

As is true with most methods in clinical chemistry, those that are short, simple and accurate to within a -5% range, are the ones most commonly selected for general use. Thus, ideally, for total cholesterol, the analysis should be made directly on the serum sample with the latter in small amount to be applicable for pediatric use, the reagents should be stable and minimal in number, and the method should be rapid, as specific as possible and apply to all types of clinical serum samples, regardless of the presence of bilirubin, hemolysis, drugs, etc.

One technic which nearly satisfies the above criteria was published in 1953 by Pearson, Stern and McGavick<sup>5</sup> and depended upon the serial addition to serum

\*From the Department of Medicine, Central Maine General Hospital, Lewiston, Maine. This was sponsored in part by the Maine Heart Association.

Table I

METHODS FOR CHOLESTEROL DETERMINATION		
Method	Principle	Comment
Schoenheimer-Sperry	Alcohol-acetone extraction with Liebermann-Burchard color	Tedious, technical Color sensitive to heat, light
Bloor	Alcohol-ether extraction with Liebermann-Burchard color	Similar to above
Schmidt-Thome, Schettler, Goebel	Cholesterol precipitated with excess digitonin, titration of latter	Faster; indirect Less accurate
Michaels, Margen Kinsell	The cholesterol-digitonide precipitate measured by turbidimetry	Turbidimetric methods less accurate
Zlatkis, Zak, Boyle	Color reaction with ferric chloride	Color not specific, interference from other chromogens
Forbes, Irving	Extraction method. Liebermann-Burchard color	Similar to first above Shorter, accurate
Abell, Levy, Brodie Kendall	Alkaline-alcohol digestion Extraction with hexane Liebermann-Burchard color	Similar to Forbes-Irving
Carr, Drechter	Acetic acid-acetic anhydride. Protein precipitate removed. Sulfuric acid added to give Liebermann-Burchard color	Shorter than first Accurate

From resumé by Hollinger, Austin, Chandler and Lansing<sup>4</sup>

of sulfuric acid, glacial acetic acid, p-toluenesulfonic acid and acetic anhydride. This method was accurate, gave reproducible results and was rapid. More recently, however, Huang, Chen, Wefler and Raftery<sup>6</sup> have reported the development of a stable cholesterol reagent based on the Liebermann-Burchard color reaction, which is added to the serum sample as a single solution. During the past year this has been adapted to a single-purpose photoelectric photometer with a dial from which one reads directly the cholesterol content of the serum sample.<sup>7</sup> In addition to the instrument, one purchases the stable cholesterol reagent, precision cuvettes, disposable pipettes, color standards and cholesterol standard control serum.

In view of the fact that this instrument and method have been widely advertised as a means of rapid, accurate serum cholesterol measurement, a check of these claims about such an important test has seemed most desirable. No other evaluation of its use and accuracy has been noted by this writer. Accordingly, it is the purpose of this paper to relate some five months' experience with this method.

#### METHOD

All serum samples reported below were obtained from the laboratory at the Central Maine General Hospital where the total cholesterol had been determined on each by duplicate analyses using the Schoenheimer-Sperry technic. Testing of each sample was accomplished by filling the special pipette with the serum, transferring this to a special cuvette and addition of the color reagent to a mark on the side of the cuvette. After incubation at 37°C. for 10 minutes, the cuvette was placed in the previously standardized colorimeter and the cholesterol content read directly on the instrument dial. The entire procedure takes less than 15 minutes. The cost of each single test, not including labor, is that of the disposable pipette and reagent, or approximately 60 cents.

#### RESULTS

Patient	This Method	Schoenheimer-Sperry	Percent Error
Con	211 mg%	205 mg%	+ 2.9%
Jac	241	205	+17.5
Smi	355	300	+18.3
Han	255	229	+11.3
Hyl	226	200	+13.0
Whi	323	303	+10.0
Bre	329	310	+ 6.1
Ras	266	256	+ 3.9
Fow	298	285	+ 4.6
Rob	424	420	+ 1.0
O'C	273	256	+ 2.7
Abe	261	237	+10.0
Jud	273	246	+11.0
Tom	290	310	- 6.4
Lav	375	406	- 7.7

Hou	231	231	0.0
Con	194	205	- 5.4
Hyl	212	200	+ 6.0
Hua	223	224	- 0.4
Hed	325	321	+ 1.2
Mar	318	318	0.0
Mil	264	269	- 1.9
Par	209	204	+ 2.4
Hal	201	192	+ 4.7
Har	184	176	+ 4.5
Gro	153	140	+ 9.3
Xan	247	239	+ 3.3
Mer	284	275	+ 3.3
Jor	261	266	- 1.9
Isa	263	269	- 2.2
Bol	287	282	+ 1.8
Bla	300	302	- 0.6
Pet	240	235	+ 2.1
Sho	240	240	0.0
Mil	341	335	+ 1.8
McN	238	243	- 2.1
Sma	250	260	- 3.8
Rem	255	252	+ 1.2
Wel	301	278	+ 8.3
Des	279	264	+ 5.7
Wel	301	278	+ 8.3
Fle	257	250	+ 2.8
Gag	212	185	+14.6
Mas	279	277	+ 7.2
Gre	256	249	+ 2.8
Gag	220	185	+ 8.1
Bar	154	146	+ 5.5
Gam	328	330	- 6.1
Abr	312	316	- 1.2
Rey	345	335	+ 3.0
Dun	312	300	+ 3.0
McC	312	329	- 5.2
Tow	201	220	- 8.6
Bor	286	280	+ 2.1
Jon	177	174	+ 1.7
Fob	262	274	- 4.4
Ste	231	227	+ 1.8
McC	299	329	- 9.1
Jon	172	164	+ 4.9
Tow	206	220	- 6.4
God	348	349	- 0.3
Buc	199	203	- 1.9
Bro	282	298	- 5.4
Bro	283	298	- 5.0
Sha	454	460	- 1.3
Mur	272	270	+ 0.7
Woo	182	190	- 4.2
Coh	263	277	- 5.4
Phi	253	237	+ 6.7
Coh	268	278	- 3.6
Phi	249	237	+ 5.1
Rus	296	296	0.0
Bry	433	435	- 0.4
Kin	221	219	+ 0.9
Tra	245	246	- 0.4
Emo	313	304	+ 3.0
Mal	194	188	+ 3.2
Sta	310	316	- 1.9



Bea	217	212	+ 2.3
God	360	330	+ 9.1
Rou	253	250	+ 1.2
Gol	189	183	+ 3.3
God	224	218	+ 2.8
McL	171	168	+ 1.8
Cho	202	205	— 1.5
Smi	233	240	— 2.9
Bla	201	186	+ 6.1
Cal	161	144	+11.8
And	203	192	+ 5.7
Her	199	186	+ 7.0
Far	200	192	+ 4.2
Dob	251	252	— 0.4
Dea	302	314	— 3.8
Ric	186	178	— 4.5
Whi	361	345	+ 4.6
Tay	264	263	+ 0.4
Hay	221	221	0.0
McC	274	281	— 2.5
Ben	281	287	— 2.1
Mel	234	244	— 4.1
Sai	301	309	— 2.1
Tra	294	311	— 5.5
Sco	329	336	— 2.1
Boo	291	305	— 3.6
McC	328	326	+ 0.6
Str	293	296	— 0.9
Fer	249	241	+ 3.3
The	540	612	—11.0
Gum	277	284	— 2.5
Roy	260	252	+ 3.2
Cra	238	230	+ 3.5
God	368	330	+11.5
Dun	330	284	+16.2
Dum	222	226	— 1.8
Hod	283	293	— 3.9
Yen	214	189	+13.2
Mar	332	348	— 4.6
But	341	368	— 7.3
Dun	305	284	+ 7.4
Mar	318	348	— 8.1
But	332	368	— 9.8
O'B	209	220	— 5.0
Gag	158	152	+10.5
Smi	192	198	— 3.0
Har	282	276	+ 2.2
Bur	268	265	+ 1.1
Jac	162	149	+ 8.7
McC	258	268	— 3.7
Law	295	319	— 7.5
McC	363	375	— 3.2
Dan	353	372	— 5.1
Mic	373	374	— 0.3
Fon	266	271	— 1.8
Cul	274	274	— 0.4
Abb	251	260	— 3.5
Mar	162	153	+ 5.9
Tur	257	260	— 1.2
Fos	266	287	— 7.3
Tat	276	272	+ 1.5
Pus	329	340	+ 3.2
Osg	243	250	— 2.8
Cre	222	216	+ 2.8

Total: 141 serum samples  
76 over-estimated — average —5.1% error  
65 under-estimated — average —3.7% error  
Mean error —4.48%

DISCUSSION

This has proved to be a very simple and rapid method for the colorimetric determination of the serum total cholesterol. In this series of tests the first few showed an appreciable percent error. This could have been due to the lack of technical familiarity with the method or it may have been the result of an altered batch of reagent. It was obvious that with the acquisition of new reagent all subsequent tests were much better. In any event, after this first short phase, results were within the acceptable -5% error range. The over-all mean error was 4.48%. Of the 141 serum samples which were tested, 76, or slightly over one half, were high, and 65 were low, representing a nearly equal division of extremes.

A few of these serum samples were mildly icteric and this was not observed to effect results. None were grossly hemolyzed so that any unfavorable effect of this on the color development was not evaluated. Many of the patients from whom the serum samples were taken were receiving various drugs such as digitalis, dicumarol, pentaerythritol tetranitrate, etc., and this feature was not observed to influence color development.

It is felt that this method is simple, rapid and fairly accurate. Even though it does not satisfy the criticisms which have been raised against similar short methods for cholesterol determination in the past, it is thought to be sufficiently accurate to justify its use as a screening procedure in clinical medicine.

SUMMARY

1. A recently developed and simplified method for the rapid determination of serum cholesterol has been evaluated in comparison with a standard method. This method is based upon the use of a single-solution reagent for production of Liebermann-Burchard color.
2. A total of 141 serum samples were tested. The over-all mean error was 4.48% with a -5.1 to -3.7% error range.
3. This method is simple, rapid, fairly accurate and inexpensive and can be recommended as a suitable laboratory screening procedure for the determination of serum total cholesterol.

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# Misuse Of Postprandial Blood Glucose Determinations

RONALD S. POTTS, M.D., C.M.\*

This is the screening procedure of choice to detect abnormal hyperglycemia.<sup>1</sup> Unfortunately it is usually referred to as the one-to-two hour postprandial blood glucose concentration and this has caused misuse. It is necessary to remember this determination is based on the peak level which might be attained during the formal oral glucose tolerance test, normally occurring within one half hour after quickly ingesting the glucose load, and not normally exceeding 150 mgs.% true blood glucose concentration.<sup>1</sup>

When a meal of at least 100 grams carbohydrate content is substituted, the challenge is spread over 15 minutes to an hour. The timed interval begins at the meals end, but some glucose has already been absorbed. One hour postprandial is about the equivalent of 1½ hours during an oral tolerance test; 2 or 2½ hours p.c. of course are even later. To be sure, an abnormal elevation 2 hours p.c. means the peak is too high and the elevation has persisted too long. The point to be appreciated is that by 2 hours p.c., many diabetics and persons with hepatic insufficiency will be able to restore blood glucose levels to less than 150 mgs.% and hence apparently have an acceptable response according to the common definition.

To illustrate this, the total 395 oral glucose tolerance tests performed at this hospital for the years 1960-1963 were analyzed. Fifty-five percent of these, or 218, were definitely indicative of glycogenetic insufficiency. All but 4.6% of the abnormal tolerances had 1 hour levels greater than 150 mgs.%. Forty-two percent of these abnormal tolerances had 2 hour true blood glucose levels less than 150 mgs.%, and hence certainly would have had acceptable 2 hour postprandial glucose levels. Only 1.1% of these latter had 1 hour levels less than 150 mgs.%.

REPRESENTATIVE ABNORMAL GLUCOSE TOLERANCES

Case	F	½ hr	1 hr	2 hr
1. Blood	77	182	172	83
	Urine	0	3+	4+
2.	89	168	198	111
	0	0	3+	3+
3.	80	171	218	204
	0	0	3+	4+
4.	68	140	246	208
	0	4+	4+	2+
5.	87	199	207	131
	0	0	3+	3+
6.	88	185	236	123
	0	1+	3+	2+
7.	65	124	167	121
	0	0	0	0
8.	81	196	197	188
	0	0	2+	2+
9.	81	176	125	80
	0	2+	4+	4+
10.	69	170	109	94
	0	2+	1+	0
11.	66	132	204	205
	0	1+	3+	0
12.	96	180	186	119
	0	1+	1+	1+
13.	84	177	173	130
	0	1+	3+	3+
14.	94	152	177	128
	0	0	1+	4+
15.	71	193	260	237
	0	0	1+	2+
16.	77	188	208	116
	0	0	1+	1+
17.	85	172	128	95
	0	1+	2+	1+
18.	89	175	207	130
	0	0	2+	1+
19.	84	192	191	146
	0	1+	4+	2+

\* Associate Pathologist, Central Maine General Hospital, Lewiston, Maine.



20.	102	142	166	120
	0	1+	3+	3+
21.	95	205	226	129
	0	0	1+	0
22.	76	133	171	132
	0	0	0	0
23.	87	153	172	88
	0	3+	3+	4+
24.	81	184	163	126
	0	0	—	—
25.	90	176	200	138
	0	—	—	—
26.	82	136	212	216
	0	0	0	4+
27.	81	143	188	123
	0	0	2+	3+
28.	87	167	176	115
	0	0	2+	3+
29.	82	99	182	116
	0	1+	2+	0
30.	64	178	234	142
	0	0	0	0
31.	77	174	188	142
	—	—	—	—
32.	74	131	160	119
	0	0	3+	1+
33.	90	324	438	297
	0	0	3+	3+
34.	84	204	196	148
	0	—	—	4+
35.	77	162	235	118
	0	—	0	3+
36.	87	155	187	120
	—	—	—	—

37.	95	174	180	102
	—	—	—	1+
38.	72	197	210	176
	0	0	1+	1+

From this same analysis, 57% of the abnormal tolerances began with normal fasting true blood glucose levels of 65-90 mgs.%. This again demonstrates the unreliability of the fasting blood glucose determination to detect abnormal hyperglycemia. Of the abnormal tolerances, 34.5% had both normal fasting and acceptable 2 hour blood glucose levels – the people with latent and mild diabetes, or hepatic disease, who have the most to gain from early detection.

Thus we see that two hours p.c. is too late an interval to reliably demonstrate abnormal hyperglycemia – the peak which may have been too high may have already occurred and homeostatic mechanisms produced an acceptable level below 150 mgs.%.

We should discard the one-to-two hour postprandial blood glucose determination as a screening procedure and instead utilize the one hour postprandial determination, realizing this is about the equivalent of 1½ hours during an oral tolerance test. There must be greater respect for remaining reserve in glycogenetic metabolisms already insufficient.

REFERENCE

1. Miller, S. E., "A Textbook of Clinical Pathology," 6th Edition, pages 255-263, Williams & Wilkins Co., Baltimore, 1960.

Annual Meeting Dates For Your 1964 Calendar

Maine Medical Association, June 14-16, 1964 at The Samoset, Rockland, Maine.

American Medical Association, June 21-25, 1964 at the Fairmont and Mark Hopkins hotels and Civic Auditorium, San Francisco, California.

# Long Term Office Anticoagulation\*

DANIEL M. SWAN, M.D.

Long term anticoagulation therapy in the treatment and prevention of thrombo-embolic disease appears to be becoming increasingly employed. Before the era of anticoagulants, Manchester<sup>1</sup> reported 60% recurrence of myocardial infarction within five years. Levine<sup>2</sup> estimated that 75% of his patients with myocardial infarction did not survive for more than five years. Bland and White<sup>3</sup> found a 75% mortality within ten years.

In contrast, Suzman<sup>4</sup> reported a series of 1,461 cases of coronary thrombosis followed from one to ten years. The mortality of the group which received continuous anticoagulation therapy was 16.8% while those who received short term anticoagulant therapy only had a mortality of 44.5%. Nichol<sup>5</sup> reported co-operative studies of ten physicians from ten different parts of the United States consisting of 1,091 patients with coronary artery disease who received long term anticoagulant therapy and in the treated group, the mortality was 6.2%. In the control group of 319 patients who abandoned treatment, the mortality was 22.2%. In another control group of 417 patients not treated with anticoagulants, the mortality was 37.4%. Wright and associates studied long term anticoagulant therapy of cerebrovascular disease. They observed a total of 57 patients with on and off anticoagulant therapy. Thromboembolic episodes in the treated group as compared with the nontreated group were approximately in the ratio of one to eight or ten. Sickert and Milliken<sup>6</sup> reported on long term anticoagulant therapy in syndromes of carotid artery and basilar artery insufficiency and they obtained apparently excellent results.

These results and experience with long term anticoagulation therapy now make it advisable for the internist or generalist to offer it to the patient if conditions for its use are satisfactory. This is so not only because of the increased longevity afforded to the patient, but also because of increased avoidance of economic disaster to families involved, and preservation of individuals valuable to society often stricken in their most productive years.

However, there are conditions which must be met before long term anticoagulant therapy can be safely employed. First this includes detailed knowledge by the physician of the action of at least one satisfactory anticoagulant drug and of the laboratory method employed in its control. The physician must be willing to devote the time necessary to all the details involved. The physician must also possess the fortitude to employ a therapeutic agent which may involve risk of complications.

\*Reprinted from the Journal of The National Medical Association, March 1963, Vol. 55, pp. 172-175.

DISEASES TREATED	
Myocardial infarction	79
Coronary insufficiency with angina pectoris	22
Cerebral thrombosis and embolism	16
Cerebral vascular insufficiency	6
Permanent atrial fibrillation	10
Atrial fibrillation with peripheral embolism	2
Chronic cardiac decompensation	2
Retinal vein thrombosis	2
Thrombophlebitis of leg with pulmonary embolism	2
Thrombophlebitis of leg	1

A suitable well equipped laboratory must be available either in the physician's office or within easy reach of all patients receiving the therapy. The laboratory must be staffed by conscientious technicians skilled in performing accurate prothrombin time determinations. The patient must be intelligent enough to comprehend details of the treatment and must be willing to cooperate in carrying it out.

Long term anticoagulant therapy has been used in 142 cases, since 1951. The average duration of treatment was 16.7 months. One hundred cases are now under active treatment. Long term anticoagulation therapy is defined as covering three months or longer.

In the coronary insufficiency group special indications for starting therapy were considered to be increasing frequency of anginal attacks, the appearance of angina decubitus, decreasing response to nitroglycerin and increasingly abnormal electrocardiograms when it was felt that myocardial infarction was impending.

Additional diseases treated by others with this therapy have included pulmonary hypertension, arteriosclerosis obliterans and among relatively rare conditions, thrombotic thrombocytopenic purpura, polycythemia, Chiari-Budd syndrome, visceral Buerger's disease, hepatic vein thrombosis and others.

Diseases generally considered to definitely contraindicate long term anticoagulant therapy are active duodenal or gastric ulcer, gastric carcinoma, other ulcerative lesions of the gastrointestinal tract, bleeding polypi, cirrhosis of the liver, severe hypertensive disease, subacute bacterial endocarditis, Meckels diverticulum, cerebral hemorrhage, cerebral aneurysm, severe renal disease, dissecting aneurysm of the aorta, hepatitis, any congenital or acquired blood dyscrasias interfering with blood clotting, polyarteritis, recent operations on the brain, eyes or spinal cord, Vitamin C deficiency as in scurvy, and late pregnancy.

Contra-indications which are considered to require caution in the use of anticoagulants are mild renal insufficiency, hypoprothrombinemia, large open



wounds, moderate hypertension, acute or chronic passive congestion of the liver, and recent operations on the urinary bladder.

*Anticoagulants employed:* — Bishydroxycoumarin (Dicumarol®) was used for several years. Use of this drug was abandoned because of the necessity of excessively rigid laboratory control, requiring frequent prothrombin time determinations involving inconvenience and increased expense to the patient as well as unpredictability of response with frequent escape from the therapeutic range. It was found unsatisfactory in general for office long term anticoagulation. Warfarin sodium (Coumadin®) has been used for the past several years and has been found to offer relative satisfactory regularity and predictability of prothrombin response and has required minimal need for estimation of the prothrombin time. A greater rapidity of action was noted with Coumadin as compared to Dicumarol. Many of the patients obtained hypoprothrombinemic effects in 24 hours and almost all obtained therapeutic effects in 36 to 48 hours. Patients receiving Dicumarol usually require 60 to 90 hours to develop therapeutic levels.

*Method:* — The one stage prothrombin time determination method of Quick employing simplastin and diagnostic plasma has been used as an office procedure and has been found rapid, relatively simple and dependable. The majority of patients were started on anticoagulation therapy in the hospital, however some were started in the office and in this case having ruled out any contra-indications, an initial prothrombin time determination is done before medication is administered. Prothrombin time determinations are done at first every one to two days, increasing to every third to fourth day, then at one, two, three and finally four weeks depending upon the ease of control. Patients who continued to receive medication following discharge from the hospital received prothrombin time tests every two, three and finally every four weeks in most cases. These intervals are shortened as required by any complication or clinical change. Details of the treatments are explained to the patient and they are informed of the risks of overdosage and underdosage. They are supplied with an instruction sheet outlining the complications to watch for and instructing them to report immediately any large bruising, melena, hematemesis, hematuria or any other signs of prolonged bleeding. They are supplied with a card to carry in their wallet stating that they are taking anticoagulant medication in case of accident. Patients are not usually detained at the office to obtain the results of the test but are instructed to continue the same dosage unless they are advised by telephone to change as the result of finding the prothrombin time level unsatisfactory on either the high or low side. The prothrombin time is maintained at one-and-one-half to two times normal as expressed in seconds, the average normal usually being 15.5 plus or minus 1.5 seconds. The range sought for has been 21 to 31 seconds. Generally speaking, no case of serious hemor-

rhage was noted when the prothrombin time was 31 seconds or less. According to the experience of the author, danger of hemorrhage increases appreciably with each second prolongation over 31. Urinalysis is performed at regular intervals. Patients are advised to take the medication at bedtime as this appears to offer a more dependable degree of absorption due to the usually empty stomach. Patients are usually supplied with Vitamin K<sub>1</sub> tablets (Mephyton®) and are instructed to take one every hour in case bleeding is noted, when they are unable to contact the physician immediately. The patient should always be supplied with Vitamin K<sub>1</sub> tablets if they are to travel out of town, or if they are likely to be unable to contact the supervising physician or a reliable substitute for any other reason. Vitamin K<sub>1</sub> tablets may be withheld from certain patients if it is felt that they are unable to refrain from taking them unnecessarily for unimportant indications. At the time of the prothrombin time test, they are given an appointment for the next test. In a majority of cases, a prothrombin time determination once a month has been found to give an adequate control. Such long intervals between prothrombin times are desirable if possible because they tend to lessen patient resistance to the therapy because of convenience of less frequent tests, fewer needle punctures and reduced costs to the patient.

For starting a patient on anticoagulant therapy in the office, the initial dose is usually 30 to 40 mg. depending on the weight and age of the patient and the level of the premedication prothrombin time. Older patients are usually started on a lower initial dose. No Coumadin is given on the second day, and the dosage thereafter is prescribed according to subsequent prothrombin tests. A hypoprothrombinemic effect is usually obtained by the second day after the loading dose. The average maintenance dosage has varied from 2.5 mg. to 12.5 mg. daily with infrequently a patient requiring more or less. No patient ever required more than 15 mg. daily. Patients are usually advised to avoid salicylates while taking anticoagulants, although in the experience of the author, 6 to 8 aspirins weekly did not significantly effect the prothrombin time in an easily regulated patient.

Many factors have been described as changing response to anticoagulant drugs. Among endogenous factors thought to cause increased response are congestive heart failure, febrile disease, renal disease, liver insufficiency, malnutrition, diarrhea, and to produce decreased response, diabetes, pancreatic malignancy, hyperlipemia, pregnancy and edema.

Exogenous factors which have been described as causing increased response include besides salicylates, antibiotics, quinidine, phenylbutazone, (Butazolidin®), decreased physical activity, diets low in protein, sulfanomides, hepatotoxic agents and among these thought to cause decreased response are alcohol, Vitamin K in multivitamins, mineral oil, corticotropine, corticosteroids, digitalis, questionably antihistamines and increased phy-

MAJOR COMPLICATIONS OF TREATMENT		Cases
Gross genito-urinary hemorrhage		11
Gross gastro-intestinal hemorrhage		5
Large subcutaneous hematoma		3
Cerebral Hemorrhage		1
Subdural Hematoma		1

RESULTS OF THERAPY				
Disease	No. of Cases	Recurrence	Deaths	Sudden Death
Myocardial Infarction	79	3	3	1
Coronary insufficiency with angina pectoris	22	2	0	0
Cerebral Thrombosis or embolism	16	0	0	0
Permanent atrial fibrillation	10	0	0	
Cerebral vascular insufficiency	6	0		
Atrial fibrillation with embolism	2	0		
Thrombophlebitis with pulmonary embolism	2	0		

sical activity. Hot weather has been found to definitely increase response.

Other factors that may cause changing response to anticoagulants are variations in absorption of the drug from the intestinal tract, Vitamin K intake in the diet varying in different seasons of the year and other unknown variables.

Coumadin therapy is stopped before all major surgery and usually minor surgery, Vitamin K<sub>1</sub> being given prior to surgery, if necessary, to obtain a normal prothrombin level before proceeding. This may or may not be done before dental work involving the risk of hemorrhage, as some dentists consider it unnecessary. Coumadin may be restarted two or three days following surgery in most cases, with Heparin being administered in the interval between if the indication for anticoagulation therapy is considered urgent.

Gross hematuria was the most frequently observed complication. Two of these patients also had renal lithiasis. One patient had both gross hematuria and on another occasion massive subcutaneous hematoma of the scapular area with drop in hematocrit of 26 following which treatment was discontinued. The hematuria yielded readily in each case to vitamin K therapy and they were usually not hospitalized. The case of cerebral hemorrhage occurred following a course of phenylbutazine for acute arthralgia and was fatal. The prothrombin time was found high on admission to the hospital. The case of subdural hematoma may or may not have been drug induced. However, the anticoagulant had been continued following the head injury because of subsidence of symptoms

with the prothrombin time being within therapeutic range. This case, too, was fatal in spite of surgical drainage of the clot. Four of the cases of gross gastro-intestinal bleeding required blood transfusions. Four of them had subsequent findings of duodenal ulcer by x-ray. The fifth was not x-rayed but had an old ulcer history. Two were again started on anti-coagulant therapy without incident to date. Two of the significant hemorrhages occurred in patients who had failed to report on time for prothrombin time tests as directed.

Minor bleeding was observed fairly frequently and consisted of epistaxis, oozing from the mouth and gums, from cuts while shaving and bruising with only slight or no trauma.

Treatment of Complications: – Vitamin K<sub>1</sub> (Mephyton) is a satisfactory specific antidote for excessive hypoprothrombinemia due to anticoagulant medication. In most cases of minor bleeding, 5 mgs. of Vitamin K<sub>1</sub> is given every hour until results are obtained. Massive hemorrhage is treated with 50 to 100 mgs. given either intravenously or by mouth and whole blood is given if necessary. Drug induced hypoprothrombinemia of dangerous degree due to Coumadin has been found easier to reverse with Vitamin K<sub>1</sub> than when produced by Dicumarol.

Patients who had sustained a major hemorrhage were generally resistant to the reinstitution of long term anticoagulant therapy. However, they were usually urged to do so except in the presence of a previous history of upper gastro-intestinal tract bleeding. In these cases, danger of recurrent hemorrhage must be weighed against the risk of the thromboembolic disease under treatment.

COMMENT

The number of cases discussed in this paper is relatively small, however they appear to confirm results obtained in larger series. Usually those patients who were receiving long term anticoagulation therapy where atherosclerosis was considered to be involved had also been placed on low cholesterol diets, and usually received vasodilators, anti-cholesterol medication and anti-hypertensive therapy if indicated. This additional therapy may somewhat becloud the issue in evaluating the above mortality results as contrasted to mortality figures of 15 to 20 years ago and also makes it difficult to accurately assess the marked improvement noted in some patients with angina pectoris. Nevertheless, these results appear to confirm the conclusion that long term anticoagulation therapy is a useful therapeutic agent in the prevention of mortality and morbidity in thrombo-embolic disease.

REFERENCES

1. Manchester, B.: The Value of Long Term AntiCoagulant Therapy, Ann. Int. Med. 47:1202 (Dec.) 1951.
2. Levine, S. A.: Clinical Heart Disease, Philadelphia, 4th Ed., 1952, W. B. Saunders Co., P. 1116.
3. Bland, E. F. and White, P. D.: Coronary Thrombosis (With



## INTERIM MEETING

Maine Medical Association House of Delegates

Sunday, April 12, 1964

THE STOWE HOUSE, BRUNSWICK, MAINE

Dinner at 12:30 P.M. followed by the Business Meeting

Presiding, Linus J. Stitham, M.D., Dover-Foxcroft

Speaker of the House of Delegates

The Order of Business will include financial statement for 1963, proposed budget for 1965 and other matters to be acted upon at the Annual Meeting in June.

Complete agenda for this meeting will be mailed to each of the county delegates and alternates.

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### Maine Medical Association Council

The Council will meet at the M.M.A. headquarters in Brunswick, Maine at 10:00 A.M.



DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

Department of Health and Welfare

Hospital And Nursing Home Care Expenditures  
Total Over \$4 Million Annually In Maine

The State's program of nursing home care for recipients of public assistance totaled \$2,846,959 in the last fiscal year, an expenditure which when added to the sum of \$1,463,590 in hospital care payments for the same categories, produced a grand total of \$4,310,549 in fiscal 1962-63. If the present rate of expenditures in these two programs continues at its present pace for the balance of the current fiscal year, it is estimated that it will total approximately \$4,500,000.

The expenditures by program, by fiscal year, July, 1957 (the year that the nursing home program was started) through December, 1963 are shown in Table I.

rate is \$190 per month. The great majority of the patients are in old age assistance. As a matter of fact, about 10% of the old age assistance caseload is in nursing homes all the time. There are no limitations on the length of time during which we will make these payments. The average length of stay in nursing homes is approximately 7 months. Patients leave nursing home status by death, removal to home or hospital, or reclassification to a different level of care. Up to the limit of the federal matching ceiling, which we are closely approaching incidentally, nursing home payments are about 2/3 federal money.

TABLE I

Fiscal Year	Hospital Care					Nursing Home Care*					Grand Total
	Old-age	Blind	Disabled	Dependent Children	Total	Old-age	Blind	Disabled	Dependent Children	Total	
1957-58	\$637,592	\$20,628	\$75,699	\$184,222	\$918,141	\$153,046	\$4,245	\$15,936	\$	\$173,227	\$1,091,368
1958-59	625,785	16,197	98,750	239,316	980,048	729,064	16,613	84,753	151	830,581	1,810,629
1959-60	669,425	15,469	117,527	263,561	1,065,982	852,450	15,931	114,700	59	983,140	2,049,122
1960-61	715,827	18,966	141,009	297,567	1,173,369	1,379,453	26,264	167,340	467	1,573,524	2,746,893
1961-62	832,166	19,139	149,436	336,574	1,337,315	2,178,524	40,271	255,690	1,136	2,475,621	3,812,936
1962-63	900,638	15,255	190,088	357,609	1,463,590	2,530,253	37,364	277,344	1,998	2,846,959	4,310,549
July-Dec. 1963	422,404	7,404	96,109	167,768	693,685	1,375,994	20,352	157,062	246	1,553,654	2,247,339

\*These amounts represent payments from the medical care accumulation fund only. Prior to February, 1961, nursing home costs are understated since the money payment to the recipient was applied to the cost of nursing home care and the difference was paid from the pooled fund. After February 1, 1961, all payments for nursing home care were paid from the pooled fund.

During the same period of time, the rate of payments to nursing homes for public assistance recipients increased from \$135 per month on July 1, 1957 to the present rate of \$190 per month, or an increase of 40%.

Excerpts from the Department's budget presentation at the special session contain numerous facts on the nursing home situation in general and may be of interest to physicians:

"Currently, we are paying in whole or in part for the care of approximately 1,750 public assistance recipients who are patients in some 150 nursing homes. The nursing homes involved for all practical purposes are all private businesses located throughout the state. They vary in size from five to fifty beds. The patients with whom we are concerned represent about one-half of all nursing home patients in the state. The individual

"Our licensing standards for nursing homes are not as stringent as they might be but probably are a reasonable compromise of all of the factors currently involved. They attempt to assure a reasonable level of quality of care, and by classification we attempt to minimize the frequency with which a patient not needing this level of care is placed in a nursing home. (We make no placements ourselves. This is left to family, friends, town officials, etc.) Our licensing standards do enforce a certain minimum cost level for nursing home operation by the requirements for staff, fire protection, etc. To some extent costs of operation can be influenced by changes in these licensing standards. There is no expectation at this time that licensing standards will become more rigorous next year.

"Obviously, the costs of operation of nursing homes



will vary from place to place and between different institutions dependent upon size, location, type of buildings, administration, wage rates, percentage of occupancy, methods of calculating costs of operation, etc. It is very difficult to get completely comparable cost data from all nursing homes. From the cost data obtainable the median patient day cost appears to fall in the \$6.50-\$7.50 range. Obviously there are individual instances where the costs may be over or under these figures. On a monthly basis these costs become \$195.00-\$225.00. As has been said above, we are paying \$190.00 per month for basic services. Extra charges may be made for medications, dressings, unusual supplies or unusual services if there is a source for such extra payment other than our program. For example, general relief is frequently billed for such extras.

"The proposed appropriation, to increase our rates of nursing home payments, was expected to be sufficient to increase our rates by 10% or approximately \$20 per month. However, it is now apparent that such an increase will probably raise our average grant beyond the federal matching level and, therefore, the suggested appropriation is not enough to guarantee a flat 10% increase in rates."

(Since preparation of the foregoing data, the Legislature, in special session, appropriated \$120,000 to be used in the 1964-65 fiscal year. This represented a cut of \$65,000 over the amount requested by the Department. This appropriation does not become available until mid-April, and the rate changes that may be made cannot be exactly determined as yet.)

## M.M.A. Joins With S.M.A.L.U. In Sponsorship Of Medic-Alert Idea

The Maine Medical Association will join with the Southern Maine Association of Life Underwriters in 1964 to promote an understanding of the importance of emergency medical identification.

"Project Life Guard" is designed to educate doctors, hospitals, the general public, police and fire departments and other interested organizations in the recognition of the MEDIC-ALERT EMBLEM. Both groups will strive to make membership in the MEDIC-ALERT FOUNDATION easily available to all persons who would benefit.



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Clyde I. Swett, M.D., Island Falls, member of the National Advisory Committee of the Medic-Alert Foundation, is Chairman of the project for the M.M.A.

Richard M. Salisbury, Portland, is Chairman of the "Project Life Guard" effort for the S.M.A.L.U.

## *Maine Heart Association Notes*



### **Dental Extractions In The Presence Of Continual Anticoagulant Therapy**

“Anticoagulant therapy is now widely employed in the treatment of thromboembolic diseases. In these situations, anticoagulants are frequently administered for long periods of time, during which the need for dental care frequently occurs. The hazards of oral surgery under conditions of drug-induced prolongation of the clotting mechanism have been subject to question.

“Many minor and major surgical procedures have been safely performed on anticoagulated patients. . . . These authors concluded that the most important factors in minimizing bleeding were skillful medical management and careful surgical technique.

“In contrast to the hazards of bleeding during anticoagulation, there is also the possible danger of abruptly withdrawing drug therapy.

. . . Chamberlain reviewed current experiences and concluded that the danger of intravascular clotting after withdrawal of drug therapy is greater than the danger of bleeding during anticoagulation, provided proper safeguards are used.

“Our experience indicates that single or multiple extractions can be safely performed on anticoagulated patients. Whatever risk may be involved in stopping anticoagulant therapy may thus be avoided. . . . Limited experience with patients on heparin therapy suggests that a similar degree of safety exists in handling their dental problems.”

---

Reference: Frank, B. W. et al. *Annals of Internal Medicine*, Volume 59, pages 911-913, December 1963.



Young Woman  
Reading a Letter

JOHANNES VERMEER  
1632-1675



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*Research in the Service of Medicine*



# Necrology

ARTHUR U. DESJARDINS, M.D.

1884-1964



DR. DESJARDINS

Arthur U. Desjardins, M.D., head of the Section of Therapeutic Radiology of the Mayo Clinic from 1920 to 1948, and a pioneer figure in the development of medical work with x-rays in the United States, died in the Community Hospital in Damariscotta, Maine, on January 15, 1964, of the effects of coronary arterial disease. Dr. Desjardins retired from the Mayo Clinic in 1949 and moved with Mrs. Desjardins to Damariscotta, near Walpole.

Dr. Desjardins was born in Waterville, Maine, on May 6, 1884, the son of Sarah Marie Mercier Desjardins and Samuel Desjardins. He studied at Montreal College from 1898 to 1900 and Saint Joseph's College in Three Rivers, Canada, in 1900 and 1901. In 1912 he received the degree of doctor of medicine from the University of Pennsylvania; in 1913 and 1914 he was in charge of the Fairfield Tuberculosis Sanatorium in Fairfield, Maine.

In 1914 Dr. Desjardins went to France, where he was associated with the American Hospital in Paris. He also was assistant surgeon to American Ambulance in Paris. In 1915 and 1916 he was first assistant surgeon to Dr. Joseph Blake at a military hospital in Ris Orangis, France.

On February 1, 1917, Dr. Desjardins was appointed a fellow in surgery in the Mayo Foundation, but he had been commissioned a captain in the Medical Reserve Corps of the Army in June of that year, and was ordered to active duty in August. He served as pathologist to various medical units in France, and was commanding officer of the Third Army Laboratory in Coblenz, Germany, in 1919. He was released to civilian life in July, 1919, with the grade of major. He continued his military interests in the Officers' Reserve Corps, in which he attained the grade of colonel in 1935.

Dr. Desjardins resumed his fellowship at the Mayo Foundation on January 1, 1920, but changed his major sequence to

roentgenology. In the summer of that year he studied radiologic physics at the University of Cambridge, England, and in October of that year he was made head of the Section of Therapeutic Radiology of the Mayo Clinic, a post he held until July 1, 1948, when he became a senior consultant. He retired from active practice at the Mayo Clinic on October 1, 1949.

Dr. Desjardins received the degree of master of science in radiology from the University of Minnesota in 1924. A year later he was appointed an instructor in radiology in the Mayo Foundation, Graduate School, University of Minnesota, and he was advanced to assistant professor in 1928, associate professor in 1933 and professor in 1936. In 1934 he was certified as a specialist in radiology by the American Board of Radiology, Inc.

Dr. Desjardins, at the beginning of his career, was interested chiefly in surgery; shortly after he returned from service in World War I, he changed his major interest to therapeutic radiology, and in this field he achieved nation-wide recognition as an authority. He contributed more than 100 papers to the medical literature, some of them in French, which he spoke and wrote fluently. He was a member of the Council on Physical Medicine of the American Medical Association for a number of years, and he also was a member of the Council on Industrial Health of that association. In 1939 he served as a consultant to the Federal Communications Commission on the problem of radio interference. He was recognized as being exceptionally proficient in photography, and was an early worker in color photography. His photographs received widespread attention and admiration.

Dr. Desjardins was a fellow of the American College of Physicians and a member of the Southern Minnesota Medical Association, the Clinical Congress of Internal Medicine, the Radiological Society of North America, the American Medical Association, the American Roentgen Ray Society, the American Radium Society, the Alumni Association of the Mayo Foundation and the Society of the Sigma Xi. He was an honorary member of the Roentgen Society of England. In 1950 the University of Minnesota conferred upon him a certificate of merit in appreciation of his long tenure of service to the university as a member of the faculty.

Dr. Desjardins was an Honorary member of the Maine Medical Association and the Lincoln-Sagadahoc County Medical Society, having received a 50-year pin in 1962. An article entitled "The Cardiovascular Effects of Ionizing Radiations" by Durwood J. Smith, M.D. was published in tribute to Dr. Desjardins whose monumental review of this subject, "Action of Roentgen Rays and Radium on the Heart and Lungs: Experimental Data and Clinical Radiotherapy," was published in 1932.

After he moved to Damariscotta in 1949, Dr. Desjardins became a consultant in radiology to the Community Hospital in that place, and was a prime force in efforts to raise funds for the purchase of x-ray equipment for the hospital. He took an active part in civic affairs in both Damariscotta and Walpole.

Dr. Desjardins was married to Miss Marie Laure Jeanne d'Argy on January 30, 1913. She died on September 14, 1924. On April 10, 1926, he was married to Miss Helen Beatrice Hardy, of Rochester. Dr. and Mrs. Desjardins had three children: Adrienne Aymard (Mrs. Edward Ackerman, of Washington, D. C., currently living in Switzerland); Paul Arnault, of New York City, N. Y.; and Vincent (deceased).



# County Society Notes

## SOMERSET

A meeting of the Somerset County Medical Society was held at the Oak Pond Restaurant in Canaan, Maine on December 17, 1963. The meeting was called to order by the President, W. Edward Jordan, Jr., M.D.

Following a business meeting, the American Medical Association film dealing with the Kerr-Mills Bill was shown.

Drs. Carlton E. Swett of Skowhegan and Bruce Trembly of Waterville were the guest speakers. Dr. Swett gave a resumé of Recent Advances in Surgery and Dr. Trembly spoke on Advances in Neurosurgery, showing slides of various neuro-surgical problems.

A buffet-style dinner was enjoyed following the meeting.

MARIAN L. STRICKLAND, M.D.  
*Secretary*

## YORK

The annual meeting of the York County Medical Society was held at the Goodall Hospital in Sanford, Maine on January 8, 1964. Twenty-nine members and four guests were present. Following a social hour and dinner, the meeting was called to order by the President, James S. Johnston, M.D.

The following officers were elected for 1964:

President, Roger J. P. Robert, M.D., Saco  
Vice-President, Alvin A. Hoffman, M.D., York  
Secretary-Treasurer, Charles W. Kinghorn, M.D., Kittery  
Delegates to the Maine Medical Association House of Delegates: Carl E. Richards, M.D., Sanford; Robert F. Ficker, M.D., Kennebunkport, and Roger J. P. Robert, M.D.  
Alternates: Melvin Bacon, M.D., Sanford; Stephen A. Cobb, M.D., Sanford and Kenneth E. Leigh, M.D., York  
Censors: Stephen A. Cobb, M.D.; Melvin Bacon, M.D.; and Willard H. Bunker, M.D., York Harbor

Ernest W. Stein, M.D., President of the Maine Medical Association, the guest speaker, was introduced by Dr. Robert. Dr. Stein gave a very interesting talk on state and national matters.

Paul S. Hill, Jr., M.D., Councilor for the First District, gave a talk on Council matters.

A committee consisting of Drs. Paul S. Hill, Jr., Carl E. Richards and Stephen A. Cobb was appointed to look into the matter of giving donations to worthy causes. Two hundred dollars was voted for the Community Child and Family Guidance Association.

CHARLES W. KINGHORN, M.D.  
*Secretary*

## HANCOCK

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on January 8, 1964.

Elizabeth E. Williamson, M.D., President, accepted the resignations of Russell G. Williamson, M.D. and Frank S. Cruickshank, Jr., M.D. as Vice-President and Secretary-Treasurer respectively. The President appointed W. Edward Thegen, M.D. to fill in the remainder of the Vice-President's term and Russell G. Williamson, M.D. the Secretary-Treasurer's term.

George O. Chase, M.D. of Bangor, presented a most humorous, informative and entertaining presentation of The Diagnosis of Anemia.

FRANK S. CRUICKSHANK, JR., M.D.  
*Secretary*

## COUNTY SOCIETIES

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## CUMBERLAND

A meeting of the Cumberland County Medical Society was held at the Eastland Motor Hotel in Portland, Maine on January 16, 1964. Special guests were Ernest W. Stein, M.D., President of the Maine Medical Association and Mrs. Stein and the members of the Woman's Auxiliary to the Cumberland County Medical Society.

The meeting was called to order by the President, Charles R. Geer, M.D., following a social hour and dinner.

Philip P. Thompson, Jr., M.D. urged members of the society to encourage the Governor and the Executive Council to reappoint Dean H. Fisher, M.D. as Commissioner of Health and Welfare for the State of Maine.

Senator Edmund S. Muskie, guest speaker, was introduced by Dr. Geer. Senator Muskie spoke on Medical Care for the Aged. Following his speech, a lively question and answer period was held with many members participating.

STANLEY B. SYLVESTER, M.D.

*Secretary*

## LINCOLN-SAGADAHOC

A regular meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on January 21, 1964, with twenty members and two guests present.

The society recommended that Francis A. Winchenbach, M.D. continue to represent the society as a member of the Health Insurance Committee of the Maine Medical Association for the next three years.

The secretary was directed to write letters of sympathy to Mrs. Arthur U. Desjardins on the recent death of Dr. Desjardins, and to Philip O. Gregory, M.D. on the recent loss of his son.

The society approved a motion that: The members of the Lincoln-Sagadahoc County Medical Society unanimously endorse the nomination of Dean H. Fisher, M.D. as Commissioner of Health and Welfare for the State of Maine and that copies of this resolution be sent to the two local Councilors of the Governor's Council, to Dr. Fisher, and to Governor Reed.

The following officers were elected for 1964:

President, Edward L. Kinder, Jr., M.D., Bath

Vice-President, Carl R. Griffin, Jr., M.D., Boothbay Harbor

Secretary-Treasurer, George W. Bostwick, M.D., Newcastle

Delegates to the Maine Medical Association House of Delegates: Ralph C. Powell, M.D., Damariscotta and John F. Andrews, M.D., Boothbay Harbor. Alternates: Mary J. Tracy, M.D., Damariscotta and Paul A. Fichtner, M.D., Bath

Censors: Samuel L. Belknap, M.D., Damariscotta; John F. Dougherty, M.D., Bath and John F. Andrews, M.D.

Alan W. Zeller, M.D. of Damariscotta was congratulated on his certification by the American Board of Surgery.

Daniel R. Shields, M.D. of Lewiston, guest speaker of the evening, spoke on Conditions of the Prostate.

GEORGE W. BOSTWICK, M.D.

*Secretary*

## New Members

### CUMBERLAND

Toffield B. J. Strach, M.D., 3 Deering St., Portland

Cornelius A. Toner, M.D., 31 Bramhall St., Portland

### HANCOCK

William D. McLarn, M.D., 50 Union St., Ellsworth

Elihu York, M.D., 2 E. Sunset Ave., Warrington, Florida, 32507



KNOX  
Ciro Alfaro, M.D., Atlantic Ave. and Sea St., Camden  
Emery B. Howard, M.D., 23A Summer St., Rockland

YORK  
Lawrence R. Hazzard, M.D., Breakfast Hill Rd., Greenland  
New Hampshire  
Ottone Renzulli, M.D., 346 Elm St., Biddeford

---

### Deceased

ANDROSCOGGIN  
Richard N. Goldman, M.D., 185 Webster St., Lewiston, February 8, 1964

CUMBERLAND  
Luther A. Brown, M.D., 157 High St., Portland, February 22, 1964

---

## News, Notes and Announcements

**Department of Health and Welfare  
Division of Maternal and Child Health  
Including Services for Crippled Children  
(By Appointment Only)**

### Orthopedic Clinics

Augusta – Augusta General Hospital  
1:00 p.m.: Apr. 23  
Bangor – Eastern Maine General Hospital  
9:00 a.m. and 1:00 p.m.: Mar. 26, May 28  
*Half-day session 1:00 p.m.: Apr. 23*  
Fort Kent – Peoples Benevolent Hospital  
10:00 a.m.: May 13  
Lewiston – Central Maine General Hospital  
9:00 a.m.: Mar. 20, Apr. 17, May 15  
Portland – Maine Medical Center  
(*In conjunction with MMC*)  
9:00 a.m.: Apr. 13, May 11  
Presque Isle – Arthur R. Gould Memorial Hospital  
9:00 a.m. and 12:30 p.m.: May 12  
Rockland – Knox County Hospital  
1:30 p.m.: May 21

### Cardiac Clinics

Bangor – Eastern Maine General Hospital  
9:00 a.m.: Apr. 10-24, May 8-29  
Portland – Maine Medical Center  
9:00 a.m.: Every Friday (holidays excepted)

### Cleft Palate Evaluation Clinics

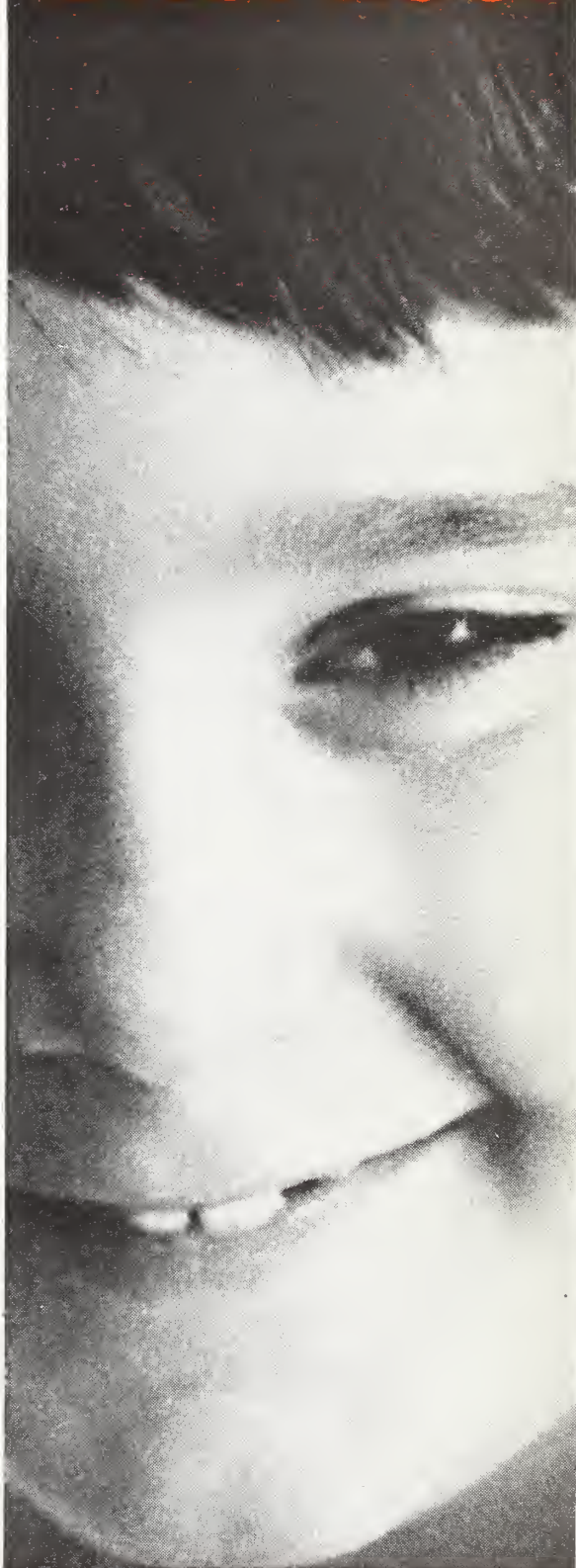
Portland – Maine Medical Center  
10:00 a.m.: May 12

### Clinics For Mentally Retarded Preschool Children

Waterville – Thayer Hospital  
9:00 a.m.: Apr. 1-15-29, May 6-20

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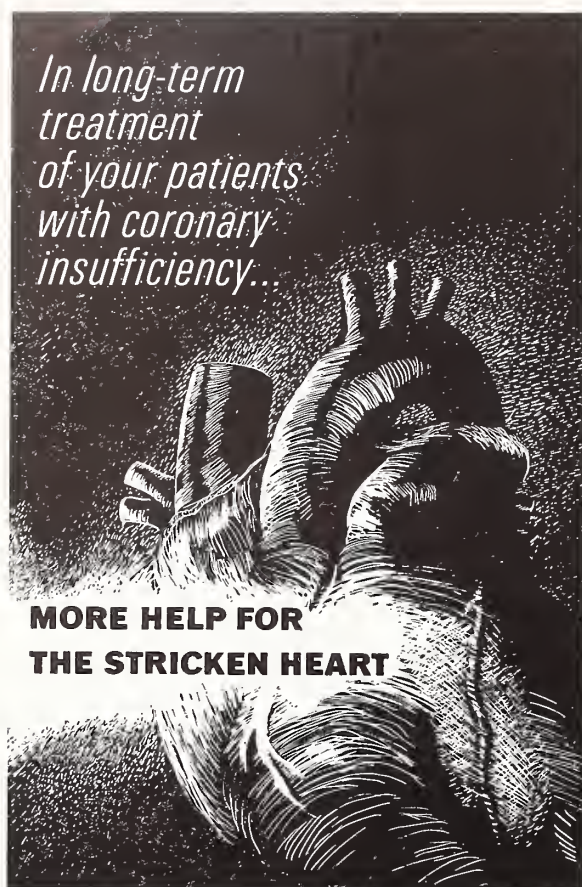
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## Book Review

**Normal Growth and Cancer** — By Grace Medes and Stanley P. Reiman. Published by J. B. Lippincott Co., Philadelphia and Montreal, 1963. Pp. 268.

This book gives a brief and lucid explanation of work now in progress in the many phases of cancer and embryology. The authors have aimed at a wide audience, assuming only a cursory acquaintance with the basic sciences. They write well and are obviously natural teachers. The subjects range over immunity and tolerance, viral carcinogenesis, chromosomes, enzymes, hormones and other host factors. New tools are outlined concisely and their evolution discussed. For example, optical instruments are traced from the earliest times to the present day, phase microscopy, ultraviolet microscopy and the electron microscope are clearly explained. Protein chemistry is covered down to the manner in which amino-acid sequences and the structure of nucleic acids were discovered. These are but a few of the subjects.

This book serves a need: without being ponderous it provides a means for the general worker — practitioner, technician, secretary or what you will — to catch up and become familiar with the most sophisticated ideas and methods of today's cancer research worker. And for the doctor who has confined himself to some narrow field and feels that the rest of the new medical world has slipped from him, that half the articles in general journals are becoming too difficult, this book will be a boon.

The remarkable thing is that the book covers such a field, yet is small enough to read as a bedside book.

ANTHONY BETTS, M.D.  
Brunswick, Maine

## LONG TERM OFFICE ANTICOAGULATION

*Continued from Page 46*

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# THE ARTHRITICS WHO COULD NOT TAKE STEROIDS

The bane of the steroids, new and old, has been the certain undesirable metabolic effects — including and water retention, edema, overstimulation of appetite, excessive weight gain, mood swings — seemed to be firmly linked to the primary anti-inflammatory action. For arthritics already overweight or with cardiovascular disease complicated by edema or those who were tense and anxious, steroid treatment could aggravate their problems. But with the advent of ARISTOCORT® Triamcinolone, many of these arthritics became "steroid-treatable." The reason: Not only did *this* steroid provide gratifying relief of inflammation and pain, but it did so *without* the penalty of overstimulation of the appetite, excessive weight gain, salt and water retention, edema, and undesirable euphoria. Six years of widespread use have confirmed these benefits for other arthritics as well as those formerly untreatable.







# The Journal of the Maine Medical Association

Volume Fifty-Five

Brunswick, Maine, April, 1964

No. 4

## MENIERE'S DISEASE

FREDERICK T. HILL, M.D.\*

This brief review of Meniere's Disease is presented because the diagnosis often is made erroneously, or, conversely, missed altogether. Thorough studies are necessary. Vertigo, generally the presenting symptom, has been described as "a confused state of the patient, often transferred to the physician."

Vertigo may be a symptom in many disorders, which often mimic Meniere's Disease such as:

- Ocular conditions, as basilar artery insufficiency
- Occlusion of anterior vertebral artery
- Pager's disease of the skull
- Brun's syndrome (cyst of 4th ventricle)
- Cerebellar pontine angle tumor
- Head injury
- Brain stem lesions
- Labyrinthitis
  - (a. secondary to suppurative otitis media
  - (b. secondary to viral infection
  - (c. toxic, drugs
- Cerebral anoxia
- Hypertension — arterio-sclerosis
- Fibrillation
- Adams-Stokes syndrome
- Carotid body syndrome
- Hyperinsulinism
- Hypometabolism
- Adrenal medullary tumor
- Petit mal
- Tetany
- Pellagra
- Drug reactions
- Postural hypotension

Fowler has reported impacted cerumen as occasionally causing vertiginous attacks, while eustachian tube obstruction may, at times, cause it.

Positional vertigo is fairly common, probably due to the action of loose utricular stauconia in the cupula of the posterior semi-circular canal ampulla.

Vertigo is such a frightening symptom to the patient that naturally it is predominant to him and may cause the physician to overlook other significant signs and symptoms. Often the physician confronted with a dizzy patient may hastily prescribe empiric therapy and, perhaps months later, refer the patient to the Otologist, who after a carefully taken history and a thorough examination, may, or may not, confirm the diagnosis; or when in doubt, seek consultation. This may suggest referral to the Ophthalmologist, the Neurologist, the Psychiatrist, or the Endocrinologist, but most often to the Internist.

### CRITERIA for DIAGNOSIS of MENIERE'S DISEASE

Recurring attacks of vertigo, generally objective — episodic — without loss of consciousness.

Low-pitched, roaring tinnitus

Feeling of fullness in involved ear

Neurisensory deafness — predominant in lower frequencies — "Cochlear deafness" — "Conductive deafness of Internal Ear" (Mygind)

Tendency to lay on unaffected ear

Discrepancy between Speech and Pure-tone Audiograms — loss in speech discrimination

Recruitment

Diplacusis — binauralis dysharmonica

Nystagmus to affected side (during acute attacks)

\*From Thayer Hospital, Waterville, Maine

Absence of temporary threshold shifts  
Hypoactive labyrinth – to caloric tests  
Nausea – often leading to vomiting  
Periods of remission

Possibly 5% may have symptoms of vertigo without deafness, or deafness without vertigo, at time of examination.

Presence of low-pitched tinnitus, diplacusis, and recruitment is of great significance in the diagnosis.

Most cases are unilateral but 5 to 10% may become bilateral.

#### WHAT IS MENIERE'S DISEASE?

Few will disagree with Williams<sup>1</sup> that it is an autonomic dysfunction or disintegration complex. It has been considered as Intrinsic allergy, Epithelial vesiculation, Herpetic neuritis. Dohlman<sup>2</sup> considers it to be on an allergic basis. Hallpike and Cairns<sup>3</sup> explain their findings of degenerative changes in the organ of Corti and stria vascularis, as due to variations of osmotic pressure in endolymph from localized vasospasm. Cawthorne<sup>4</sup> states there is a transient and reversible reaction in hair cells to chemico-physical disturbances in the endolymph. Lermoyez<sup>5</sup> defines this as circulatory dysfunction with spasm of internal auditory artery. He explains the symptoms of cochlear deafness with rapid improvement following sudden attacks of vertigo as sudden release of long-continued vasospasm of the internal auditory artery, with labyrinthine ischemia.

As a result of beautiful histopathological studies Schuknecht<sup>6</sup> has demonstrated endolymphatic hydrops with herniations, ruptures, fistulae, and collapse of the membranous labyrinth. He feels that ruptures explain the episodic nature with sudden onset of vertigo. He states that hearing loss and depression of vestibular response result from chemical alterations in the endolymph, or fine structural alterations affecting the mechanical action of the same organs; not the result of degeneration of hair cells or ganglia. He feels that endolymph accumulations occur slowly and the balance between secretion and resorption has been disturbed, and that an incompetent utriculoendolymphatic valve may be of significance.

Lawrence,<sup>7</sup> from his studies, agrees that the membranous labyrinth is capable of quick recovery by new membrane formation.

Williams<sup>8</sup> defines the histo-pathological picture as "dilatation of the scala media, ballooning of Reisner's membrane, degeneration of Corti's organ and increased mucoid elements in the perilymph, – associated with vasospasm of the precapillary arterioles and the sphincters of the stria vascularis, the source of endolymph, with resulting anoxia." The cells of Corti's organ translate the entering acoustic force into electrical energy of the active current of the neuro-fibril of the auditory nerve. The source of the required energy is the oxidative phosphorylation of glucose by enzyme systems which requires vitamins of B complex. His hypothesis, then, is

localized autonomic dystonia with sudden vasospasm or vasodilation. This was first suggested by Shambaugh in 1909.

It is generally agreed that about 5% of cases may present symptoms of vertigo without deafness, or deafness without vertigo, probably due to occlusive spasm of either the vestibular or the cochlear branch of the internal auditory artery.

It is not uncommon to see cases which do not fit into the clinical picture of Meniere's Disease or even Pseudo-Meniere's, despite thorough examination; but who may exhibit the classical findings when seen years later. This emphasizes the importance of Day's statement that the presence of the typical tinnitus – roaring low-pitched, diplacusis, and recruitment indicates incipient Meniere's and the need for early therapy. With these absent and no other explained cause found by multi-disciplinary approach, such cases should be carefully followed.

#### TREATMENT

In a large majority of cases symptoms can be alleviated by conservative medical therapy. So far at least, surgical treatment has been destructive. It has to be if it is to be effective. The only alternative would seem to be the endolymphatic shunt to subarachnoid space operation of William House,<sup>9</sup> based upon Portmann's<sup>10</sup> earlier destruction of the endolymphatic sac.

Other surgical procedures may be listed as follows:

Section of the VIII nerve (intracranial operation of Dandy).

Day's electro-coagulation of the labyrinth through the horizontal canal now superseded by his complete labyrinthectomy through the dome of the vestibule.

Cawthorne's evulsion of the utricle – first the canal approach, later via the oval window.

Schuknecht's intratympanic decompression.

(Dr. Crockett's stapedectomy approach was reported in 1903 but, unfortunately, soon forgotten.)

Ultrasonic sound.

It would seem obvious that unless medical treatment completely failed to the point of threatened suicide, or of complete incapacity on the part of a patient with unilateral deafness, there is little justification for surgery, at least on present-day knowledge. And even here psychotherapy might be given a chance.

In 1931 Mygend and Dederding<sup>11</sup> suggested what was probably the first successful treatment of Meniere's Disease. This restricted fluid daily intake to 700 cc, together with limitation of sodium chloride. Furstenberg<sup>12</sup> and his associates felt that retention of sodium was the most important factor and advised a salt-free diet, plus ammonium chloride gm. 3 tid. Talbot and Brown considered a high intake of potassium more important than restriction of sodium and advised 6 to 10 gm of potassium chloride daily.

Pilocarpine sweats, intravenous injections of histamine, and vitamin therapy all have had their advocates, with equivocal results. Recently Williams<sup>13</sup> has report-



ed a study on the effect of eriodictyal (lemon bio-flavonoid extract) on 122 patients with Meniere's disease with apparent beneficial effect as shown on the sensori-neural hearing loss. The natural history of this disease with its episodic characteristics makes careful evaluation exceedingly difficult. We have used this in a small number of cases with negative results.

Acute attacks may be controlled by atropine 1/75 gr. subcutaneously. Ephedrine, amphetamine, and epinephrine all have been reported as effective in relieving the acute attack.

Prohibition of smoking would seem indicated, as the use tends to produce vasospasm.

From all evidence pointing to Meniere's Disease as a disorder of the vasomotor system, it seems that the predominant signs and symptoms emanating from the endolymphatic system may well be due to its minute anatomical structure, enclosed within the osseous labyrinth; i.e., localization therein of a general circulatory disorder. Parsecius<sup>14</sup> in 1924 by capillary biomicroscopy, demonstrated changes in the capillaries of the nailbeds and of the lips, in the nature of deformed twisted capillary loops, in cases of glaucoma and of Meniere's Disease. The blood was flowing slowly, with evidence of sludging and even stasis. Similar changes were noted over the entire body. Because of this, and because of the many conditions mimicking Meniere's Disease and the errors often inherent in solo management, it is our feeling that a multi-disciplinary approach is desirable often with medical treatment in the hands of the Internist. With diagnosis established, generally this is in the nature of vasodilatation by nicotinic acid, or one of its compounds, plus autonomic blocking agents, such as Pro-Banthine.<sup>®</sup> Obviously this requires the Internist to be, not only interested and qualified in vascular disease, but conversant with the literature pertaining to Meniere's Disease.

## CONCLUSIONS

1. Many cases, referred with this diagnosis, after thorough study cannot be so classified.

2. Certain of these cases who at time of examination do not present a classical picture of Meniere's Disease, may eventually do so, when seen perhaps years later.

3. Meniere's Disease is a *local* manifestation, an otologic impression of a *general* vasospastic disorder, with the presenting signs and symptoms due to the minute delicate endolymphatic system being incarcerated in the dense bony structure of the petrous pyramid.

4. A multi-disciplinary approach generally is of value, especially in medical management.

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## THOUGHTS ABOUT ANTIBIOTIC COMBINATIONS

In looking at combinations of antibiotics we have seen the contributions made by following scientific curiosity. We have seen how this curiosity can be directed so that it leads to applications in disease. We have seen that these applications have to be measured by what they will do for man. We have seen that some pass the test and are widely used, whereas others fail to pass or are poorly measured. We have seen that the combined will of doctors can enforce the results of the measurement. We have seen that such leadership is desired by the majority of pharmaceutical manufacturers and that when it is given, they will respond to it. — Harry F. Dowling, M.D., in *Transactions & Studies of the College of Physicians of Philadelphia*, July 1963.

# Cost Of Medical Care

PHILIP P. THOMPSON, JR., M.D.

## INTRODUCTION

Health is a precious commodity. Illness which can be cured, most people will pay any amount to be rid of it. Illness which cannot be cured, some will pay even more for the hope of being rid of it.

The prevention of illness and the maintenance of health are as worthwhile expenditures as one finds in his yearly budget. Yet, there are few healthy people who realize this. Those who are sick either regret not spending more in preventing the illness or on recovery resent spending so much during their illness. This is the nature of the human.

However, in the past five years, the American public has shown good sense by spending more in preventive medical measures by increasing its expenditures on children under 6 years of age by 71% and by spending 73% more on the people 65 years and over. In these age groups, the preventive health expenditures pay their greatest returns.

Sixteen and one half billion dollars a year is the health bill of the American families. It has increased 6 billion dollars in the period of 5 years between 1953 and 1958 and will continue to increase. This increase represents a 42% increase in cost of services such as hospital costs, drugs, and physicians' services, but mostly it is due to the increased use of existing services. This reflects the better education of the public in health measures. Medical successes, the press, radio, TV and even, I suppose, Dr. Kildare have helped to remove the "fear" element which prevented some people from seeking medical advice.

This 16.5 billion dollars represents 5.6% of the American families' expenditures. It does represent a mean average family expenditure of \$294, but very poorly reflects the median expenditure which is \$166. In other words, wide variations in expenditures for health purposes exist. As an example, 30% spent less than \$100, another 30% spent less than \$200 while at the other end of the scale, 10% of the families had 40% of the health costs and 50% of the families bore 88% of the medical costs of the country at large.

These figures are interesting. They can be explained by several factors. The first and perhaps most obvious is the demand for "luxury care" such as King Saud demanded at a Boston Hospital a year or so ago. This is likely to be sought not only by the wealthy, but by people of moderate means in hopeless situations or in situations where there is a "ray of hope." This involves prolonged hospital stays, expensive treatments, complicated surgery, and expensive drugs. Such an illness

might cost a family \$5,000-\$15,000 a year. This of course will affect the averages.

Another facet of the same problem is genetic factors. Some families seem to have most of the "poor health" genes. In a recent survey of deaths under 50 years of age at the Maine Medical Center, it was found that almost 90% of those deaths were due to poor genetic traits. Such families have more illnesses, more serious illnesses, and more members are affected.

A third factor is the increasing percentage of the population living beyond the age of 65 years.

The complex features of the illness of the older people have inherent expenses not present among young families. Among the things that increase the expense are: (1) length of illness - Some people with cerebral vascular diseases may be completely dependent upon round-the-clock nursing care for 5-10 years or more. (2) need for hospitalization - The fact that the patient may be widowed or his spouse is not able to care for him makes care away from home necessary, and thus more expensive. (3) need for expensive treatment - Often in the case of cancer or prostate enlargement, the need for surgery is apparent. The use of oxygen tents, expensive medicines, x-ray, and other diagnostic tests increase as "degenerative diseases" occur.

The last item in the category of "special high costs" in medicine is the one which will shoot the cost of medical care up even faster in the next 5-10 year period. This is the specialized new technique used particularly in heart and vascular surgery, but also in the replacement of diseased organs. These advances are spectacular, but again "Dr. Kildare" helps to make every community demand that this service be available in his own back yard. Up goes the cost 100 times for a hundred hospitals to have such a service whereas in Great Britain or Sweden, only one hospital in the country would do such work.

At the outset, it is apparent that medical care is a complex matter reflecting the vagaries of the social science which it is. It is subject to the greatest number of variables, not the least of which may be articles in the Ladies Home Journal or Dr. Kildare's latest exploit.

It must also be stated that a large portion of the cost of medical care in our society today has no relation to actual or potentially morbid or mortal states. In other words, much of the money spent is to allay fears of disease, inquire about nuisance symptoms, or seek help for social, family, or economic problems beyond the control of the patient.

## COMPONENTS

The five major components of medical care consist



of (1) physicians services, (2) hospital care, (3) drugs and medications, (4) dental services, and (5) other medical goods and services. They divide the "medical care dollar" as follows: physicians 34%, hospitals 23%, drugs 20%, dentists 15% and other 8%.

### Physicians' Services

Five and one half billion dollars a year was paid to physicians for their services. Of this, 20% was paid for surgical services, another 10% for obstetrical services, and another 2% for eye care, while the remaining 68% was for other physicians' services.

This included office visits, hospital visits, and home visits.

This of course is gross income of physicians. Twenty to fifty percent of physicians' gross goes to cost of office, etc. His net before taxes is 50-80% of the gross.

The quantity, quality, and availability, as well as the cost of these services vary widely from the most rural to most industrialized areas of our country. Just as the availability of physicians vary, so over the country does the quality of and the demand for their services vary.

As an example, there are very few things that would make a Maine guide travel 10 miles to see the doctor while there are quite a few things which might make a suburban socialite travel to her doctor or her children's doctor during the year.

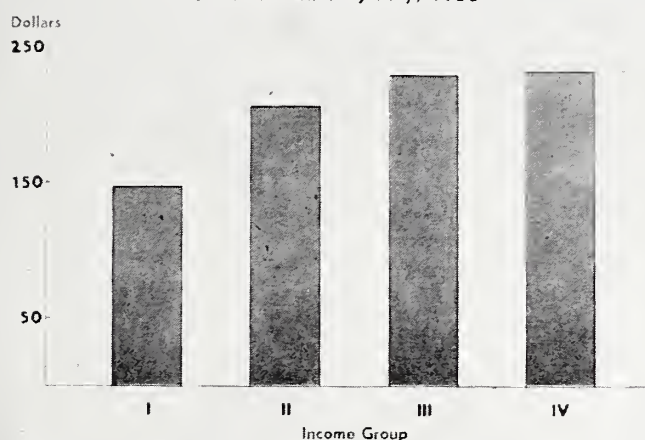
The physician's office was the place he saw three-fourths of his patients. But, the hospital and house were about equally divided as to frequency of the rest of his patient visits.

Not included in the expenditures for medical care are physicians' services provided in the accident ward and in the clinic. Here the physicians see patients and supervise their care by the hospital house staff with no remuneration. Country-wide this amounts to \$65 million of free care.

Since World War II, the number of visits to clinics and accident wards in every general hospital in the country have increased many fold. It has become a technique used by patients and doctors alike. If a family member is sick and needs attention — instead of leaving work and losing part of a days pay, the father waits until he gets home — takes the ill member to the Emergency Division where he gets good care, no medical fee, and the waiting is not time out of his weeks pay. He saves double, but may lose in the long run by not having continuity of care by one physician. In certain illnesses, this may be very important.

In the year 1962, the number of patient visits in the emergency ward at the Maine Medical Center was over 27,000 with about one-fifth being true emergencies. During the same period, there were 36,000 seen in the various outpatient clinics. A large portion of the

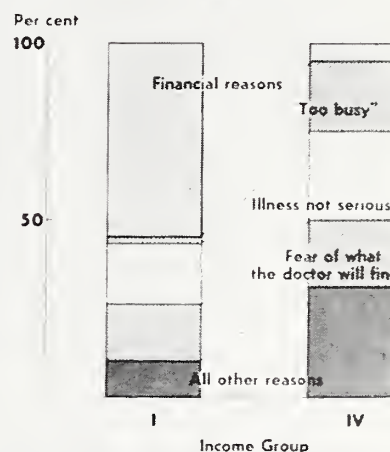
Annual Family Expenditures\* for Health Care  
Median Average by Income Group  
Selected Families, Area of Hackensack, N. J.  
Interviewed February-May, 1956



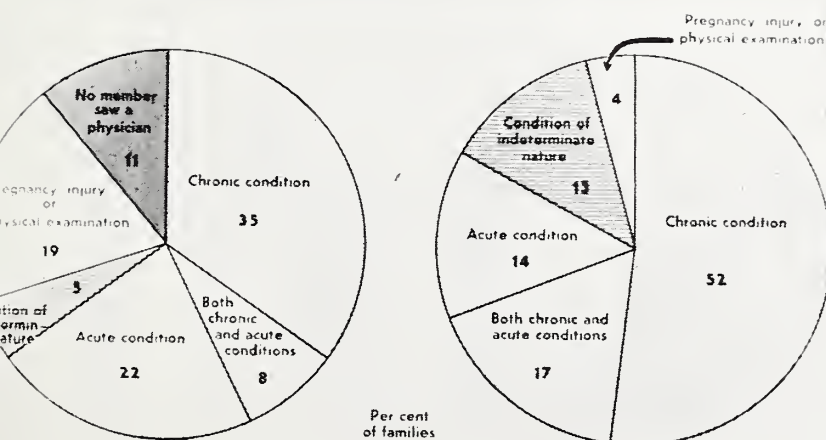
Range of annual family incomes by group: I—\$2,000 to \$5,000; II—\$5,000 to \$6,500; III—\$6,500 to \$9,000; IV—\$9,000 and over.

\*Out-of-pocket expenditures only and not including health insurance premiums. Also, includes only families who incurred expenditures during the previous 12 month period.

Reasons for Not Seeking Care\*  
Among Lowest and Highest Income Families  
Selected Families, Area of Hackensack, N. J.  
Interviewed February-May, 1956



Reasons for Seeing a Physician by Expenditures for Health\*  
United States, 1957-58

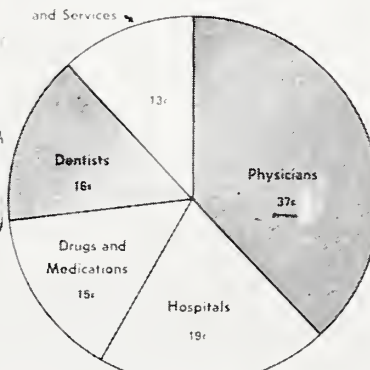


Per cent of families

Families spending \$1,000 and over

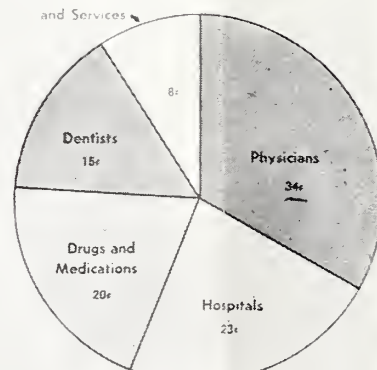
Composition of the Medical Care Dollar\*  
United States, 1952-53 and 1957-58

Other Medical Goods and Services



1952-53

Other Medical Goods and Services



1957-58

\*Distribution of reasons why family member with most severe condition(s) saw a physician.  
\*Annual personal consumption expenditures per family for all personal health services.

clinic patients are 65 years old and over, but this also included 5,200 children visits. A large portion of the emergency ward visits are children.

As suggested, the public habit of using the emergency ward of hospitals is aided by physicians themselves. Prior to World War II, most physicians had evening office hours, but this is a nicety which only a few of the general practitioners continue in most communities.

Physician services are three general types – preventative, reparative, and consultative. The so called “check-up,” immunization, and prenatal visits are in the preventative category. To be most useful to the physician as well as the patient, it should include the medical biography of at least four grandparents. The knowledge of genetics and its immediate importance to any one individual is better understood and appreciated in the past five years. Long range preventative health programs can only be planned with this genetic potential of the patient at hand. More and more physician visits in the future will be of this nature.

As a matter of interest, the much publicized British system of medical care has practically no time for this sort of medical care except massive public health immunizations. It is a luxury item that the standard of living in this country can alone afford.

Reparative medicine still comprises 90% of all visits to the physician, particularly by men. Women are much more conscious of the need for “periodic check-ups” and

preventative visits. There is one practical reason for this – the cancer screening test which is available only to women. They do see to it that their children are immunized against the various contagious diseases for which there is now protection.

It must be pointed out that the common reasons given for not visiting a physician when a symptom or sign of illness occurs (these reasons given most often by patients) are (1) hope that the illness will disappear by itself, (2) fear that the illness will be more serious or that cancer or TB will be discovered, (3) that hospitalization will be necessary, (4) that the illness will interfere with business or other usual or specially planned trips or vacations, (5) fear that disease will be labeled imaginary or psychoneurotic, (6) simple failure to recognize or believe that illness exists, (7) cannot get an appointment at a time convenient to the patient, (8) pride and cost of care.

It should not be necessary to point out the most basic of all facts pertaining to medical care. This is the need for having a family physician and becoming acquainted with him and give him time to be thoroughly acquainted with you, your past illnesses, your present illnesses, and your work as well as play habits. Whenever anything out of the normal course of events happens to your health, you should call him immediately and talk the problem over with him. He can usually tell whether it is potentially serious or not, what you

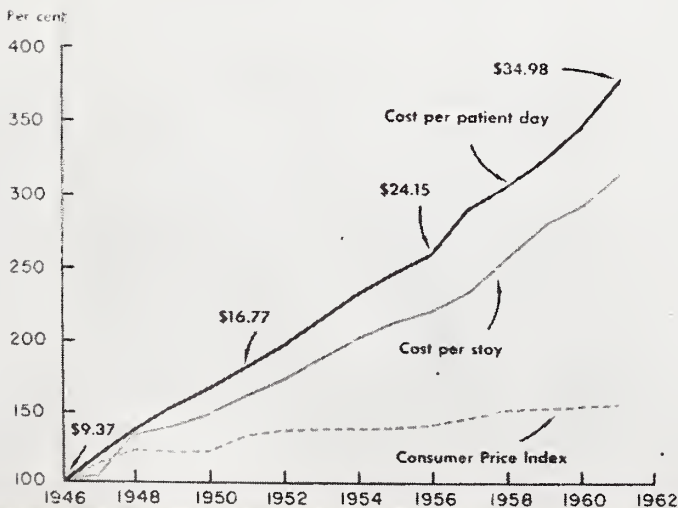
Average length of stay, average occupancy rates by bed size and per cent of beds for short-term general and other special hospitals, 1946, 1948, and 1961

Bed size category	Average length of stay		Average occupancy rate		Per cent of beds	
	1948	1961	1946	1961	1946	1961
49 and under	6.0 days	5.7 days	60.1%	59.4%	11.0%	9.3%
50-99	7.0	6.4	68.6	65.8	15.1	14.2
100 and over	9.7	8.2	74.5	77.7	73.8	76.5
					99.9%*	100.0%

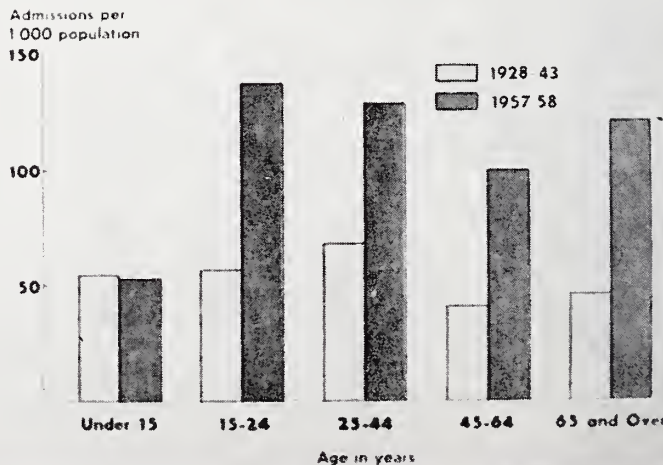
Percentage distribution of hospital charges by types of charge, and percentage increase in per diem charges: Federal Postal Hospital Association, 1947-1959

Items of hospital charges	1947	1959	% increase 1947-1959
Total	100.0	100.0	168
Room and board	51.3	47.6	150
All other	48.7	52.4	188
Laboratory	7.3	10.1	259
X ray	6.9	9.0	258
Operating room	8.5	8.1	146
Anesthetics	4.8	6.1	233
Medicines	12.8	13.2	180
Miscellaneous	8.4	5.9	—

Gross Per Cent Rise in Hospital Costs for Short-term General and Other Special Hospitals, United States, 1946-1961



Admissions to General Hospitals by Age United States, 1928-43 and 1957-58





should do about it and perform any diagnostic tests which may be indicated at the time.

*Hospital Care*

The largest single item of the total medical bill, within recent years, has been hospital care. The total has increased from 2 billion in 1953 to 3.7 billion in 1958 or an increase of 85%. Its share of the total cost of medical care has increased from 19 to 23%. This increase was caused by a 34% rise in prices and 26% increase in usage of hospital services. This amounts to an increase of 275% in the past 15 years from \$9.4 per day in 1946 to \$35 per day in 1960.

A major factor behind the rise in hospital costs is the expansion in range and volume of services provided. There is a greater number of procedures per patient day and an increase in the ancillary services offered each patient. Thus, the proportion of hospital charges for board and room has diminished while the portion assigned to laboratory, x-ray charges and medicines has increased. In general, the hospitals with the most comprehensive services have the highest costs. As an example of the capital outlay for these expanded services, the total assets per bed in 1947 was \$7,400, and has increased to \$17,800 in 1961. In the larger (more than 500 bed) hospitals, these assets per bed was as high as \$27,000.

Along with the increase in equipment was a parallel increase in number of employees increasing from 1.48 per patient in 1946 to 2.35 per patient in 1961. This represents 66% of the hospital expenses. It should be pointed out that the hospitals employ proportionately more (56%) professional and skilled personnel than the rest of the economy (11.5%).

As an index of efficiency, the length of hospital stay is a reliable guide. This has been decreasing gradually, being 5.7 days in the smaller hospitals and 8.2 days in the larger. There has been more than a day per stay decrease in the last 12 years. By decreasing the length of stay, the total hospital bill has not gone up as it otherwise would – representing a saving of \$50 in the

average bill. This average hospital bill still amounts to a considerable sum – \$267.

During an 11-year period, hospital costs have gone up 278%. Medical care costs 134%, while expenditures for education in school have increased 123%.

*Drugs and Medications*

Just as there is a wide variation in individual family expenditures for hospital, physician services, and dental care, so too is there a variation in expenditures for drugs.

- 10% spent \$0
- 18% spent \$1-19
- 21% spent \$20-49
- 22% spent \$50-99
- 19% spent \$100-199
- 10% spent \$200+

The average amount spent for drugs was \$50 a year per family by those individuals having comprehensive health insurance HIP or GHI coverage in New York City.

CONCLUSION

The providing and financing of medical care is a social problem. It is a socio-economic-medical complex. This has become a series of integrated relationships between (1) the patient – his personal habits and desires, (2) personal, public, and corporation's financial plans and budgets, (3) doctors, hospitals, pharmacists, and other vendors of medical service, (4) various third-party underwriters.

It has taken decades to arrive at this present state of integration and its resulting high quality of medical care. Such a complex does not adjust itself easily to radical changes without deterioration in quality of care. This high quality is the ultimate goal of any system of health care.

Evolutionary, slow adjustments by trial and error via the democratic processes must be made. This slow evolution according to our democratic traditions must not be replaced by the revolutionary "quick magic formulas" of the political well-doer.

Annual Meeting Dates For Your 1964 Calendar

Maine Medical Association, June 14-16, 1964 at The Samoset, Rockland, Maine.

American Medical Association, June 21-25, 1964 at the Fairmont and Mark Hopkins hotels and Civic Auditorium, San Francisco, Calif.

# Clinical Diagnosis And Management Of Hepatitis Patients\*

HEINZ F. EICHENWALD, M.D.\*\*† and HENRY R. SHINEFIELD, M.D.\*\*‡

In any discussion of a specific disease, it would seem appropriate to define the illness in question. This is relatively easily accomplished with almost every infectious illness, however, it has proven singularly difficult to develop a satisfactory definition of viral hepatitis. Perhaps the best available definition states that viral hepatitis consists of those forms of hepatitis caused by two or more hepatotropic, filtrable infectious agents, not yet identifiable by specific serologic methods, which produce systemic disease in man accompanied often, but not always, by a characteristic type of liver injury. Thus, hepatitis as an etiologic entity is definitively recognizable only by its effects on the liver, a problem complicated by the fact that many individuals infected by the hepatitis viruses show no clinically evident manifestations of liver damage. Therefore, from a public health standpoint, we may on occasion be faced with a fairly large number of individuals who are suspected of having this disease, but in whom the final proof is lacking. Outbreaks have been described where the largest proportion of the afflicted adult population demonstrated primarily gastrointestinal symptoms and children showed respiratory symptomatology, only the minority had specific signs pointing to liver disease. This is a point one must remember when one is faced with an epidemic of an ill-defined, febrile disease affecting all ages but most severely the adult, and whose clinical signs are those of an acute gastrointestinal upset. An epidemic of this type assumes particular significance if it occurs at a time when there are occasional cases of frank icteric hepatitis in the community.

How can one sharpen one's suspicions and diagnose infection with one of the hepatitis agents in the absence of readily apparent liver involvement? Experience has indicated that these diseases follow reasonably specific patterns, producing symptoms and signs in adults that are sufficiently unique to permit diagnosis with a high degree of accuracy, even in the absence of any complicated laboratory procedures. In children, the mild, anicteric illness is too variable to offer any concrete leads pointing to the diagnosis.

Let us then discuss the typical non-icteric disease in the adult. If the illness in question is infectious hepatitis, the patient will usually give a history of having felt excessively fatigued for a period of about a week. Then, he will rather abruptly develop an unusually severe headache and spike a temperature reaching as high as 103° degrees, often accompanied by chilly sensations and rarely by a frank chill. Aside from mild scratchiness of the throat and occasional cervical adenopathy, he will not demonstrate symptoms or signs referable to the respiratory tract. Shortly after the temperature elevation is first noted, the patient, if he is a smoker, will lose the urge to smoke, and will almost invariably stop this habit entirely. In addition, if he has been taking any alcoholic beverage within this time period, he will note that even small amounts of alcohol will cause a marked and for him unpredictable effect, and that a hangover will usually follow the intake of even a modest amount of alcohol.

Just following or accompanying the fever, the patient notices a progressive loss of appetite to specific foods especially those that are heavily spiced or contain a large quantity of fats derived from plant sources, such as shortening. The patient will complain of feeling excessively gassy at this point, and may experience several mild bouts of diarrhea with or without vomiting, alternating with constipation. Within 3 to 6 days of the onset of fever, the temperature will usually return to normal, and about this time, the patient will begin to experience an increase in fatigability of a quality difficult to describe but more severe than that found with other mild infectious diseases. Most individuals describe it as "I feel as if my engine had stopped," and they develop a general lack of interest and apathy, plus a type of irritability which might best be called "general crabbiness." The crabbiness is much more pronounced in women than in men, and is often so striking to permit the diagnosis of hepatitis without much further ado. Parenthetically, it might be added, that in one area where we studied an outbreak of adult hepatitis, a considerable increase in the divorce rate was reported within the year after the disease had occurred. It was our opinion that this was due to the generalized crabbiness of the population.

Women show a number of additional signs and symptoms which strongly suggest the diagnosis of hepatitis. If the infection strikes at a time just prior to the onset of a menstrual cycle, it is common for this period to be abnormal. This may mean either severe and in-

\*Presented, in part, at an American Academy of General Practice Meeting, Sanford, Maine, December 7, 1963.

\*\*Supported by National Institute of Allergy and Infectious Disease Grants CC-00088, E-3980, and AIK-64467.

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‡Assistant Professor of Pediatrics, Cornell University Medical College.



capacitating cramps with minimal bleeding, or prolonged and excessive bleeding in a person in whom this normally does not occur. It is of interest that the dysmenorrhea may continue to appear for the next several menstrual cycles, which also may be abnormal in other ways. Women will note that they are developing acne-like rashes confined chiefly to the face, they will note excessive oiliness of the skin, and the fact that hair loses its normal luster, permanent waves do not take and even setting the hair becomes impossible.

All the events that I have enumerated occur with serum hepatitis as well as infectious hepatitis, except for the fact that the serum disease is not usually marked by a temperature elevation above  $101^{\circ}$ . Additionally, in this disease the complaint of fatigue may be present for several weeks prior to the onset of gastrointestinal symptoms, and migratory joint pain and swelling occur with some frequency very early.

It is of importance to remember that all these signs and symptoms occur with great consistency in patients who demonstrate no jaundice. As with so many other things in medicine, nothing is as important as a good history, because physical examination in these mildly involved patients is often negative particularly if it is performed early in the course of the illness. Occasionally liver tenderness can be illicitly by heavily percussing the lower rib cage. A patient who has liver involvement will complain not only of discomfort following this procedure but will also usually become nauseated. Within 10 to 15 days of onset, the liver may be sufficiently enlarged to be palpable, but this does not invariably happen. Tenderness always precedes enlargement.

#### LABORATORY TESTS IN DIAGNOSIS

A relatively simple laboratory test will be abnormal even in these mild cases. This is the urine urobilinogen examination. With a little bit of practice it becomes easy to detect an abnormal intensity of the red color which develops following the addition of Ehrlich's reagent to a small urine sample. The only equipment necessary is a test tube with a mark at the 2.5 ml. level, and a dropper or pipette also with a mark indicating a volume of 2.5 ml. The test consists simply of placing 2.5 ml. of urine in the test tube, adding 2.5 ml. of Ehrlich's reagent and then 5 ml. of a solution of sodium acetate. A color develops within a minute or two, and its intensity is proportional to the amount of urobilinogen excreted.

The most sensitive test in the early phase of a mild non-icteric hepatitis is the serum transaminase determination. Serum transaminase levels in early hepatitis are always elevated, and a diagnosis of this disease cannot be sustained in the absence of a marked rise in the enzyme. The lowest level that we have observed in proven cases of hepatitis have been around 200 units; the usual increase is in the range of 500 to 2,000. BSP excretion is usually impaired quite early also, often already in the pre-icteric period but in early

hepatitis this particular determination is not as reliable an indication of liver inflammation as the transaminase level. The flocculation tests show too variable trends to be useful for the diagnosis of early hepatitis.

#### THERAPY

It should be emphasized that there is no specific therapy for hepatitis. Unfortunately, in the United States as a whole, most cases of this disease are woefully overtreated, something which is not only expensive, but often does the patient physical harm. For almost all cases of hepatitis, treatment should consist only of rest and an adequate caloric intake. Rest means bed-rest, as complete as possible. Caloric intake means calories provided in any form in which the patient prefers to take them, excluding only alcoholic beverages. The appetite is often fickle, if the patient wants to eat pickles all day, give him pickles. There is no evidence that special diets are of any benefit. Patients with hepatitis can usually eat more at breakfast than at any other time of the day. It is therefore worthwhile to reverse the usual sequence of meals and to serve them their dinner shortly after they awake in the morning. No drugs of any sort should be used for symptomatic relief of anorexia, nausea, vomiting, irritability or malaise. Almost every pharmacologic agent useful for these purposes has proven toxic to liver tissue. Tranquilizers and antiemetics should particularly be avoided. If the patient has considerable pain from dysmenorrhea the safest analgesic to use is Demerol®, Pyribenzamine® often effectively controls the nausea. Gamma globulin is useless in treatment of this disease, liver extract injections and antibiotics are contraindicated, vitamin supplementation of the diet is worthless, and to employ corticosteroid therapy in the average mild or even moderately severe case of hepatitis is, to say the least, poor medical practice.

When is steroid therapy indicated? This is a problem which has never been adequately resolved. While some investigators accept the fact that the use of steroids in precomatose and comatose cases of hepatitis has proven life-saving, other, equally reliable researchers feel that no lives have been saved by the administration of these hormones. It is true that the steroids act as cholagogues, the bilirubin level falls shortly after the initiation of therapy. We are obviously not interested in treating the patient's jaundice, but we are concerned with preventing liver damage and its long-term consequences. Then, too, the use of these compounds results in a rapid initial improvement in such subjective complaints as anorexia and malaise but again without benefiting the liver inflammation. I believe the only indication for steroid therapy is in the comatose or precomatose individual, although it must be granted that no statistically valid evidence exists that even here benefit is achieved by the administration of these agents. The reason that the steroids should not be used in the ordinary case of hepatitis despite the subjective

improvement obtained is that the treated patients demonstrated a marked increase in the incidence of prolonged and chronic hepatitis, as well as in the incidence of relapses. Relapses are far more dangerous to the ultimate well being of the patient than the initial attack itself.

In the precomatose or comatose patient, other therapeutic measures may prove to be of benefit. When symptoms such as marked lethargy develop, the protein content of the diet should be reduced temporarily, since there is adequate evidence to indicate that excessive protein may be deleterious at this time.

The administration of oral Neomycin® to suppress intestinal bacterial flora also appears to be of benefit. If the patient develops signs of bleeding, and the plasma prothrombin level is impaired, vitamin K-1 oxide, not menadione, is administered. There is no need to give this vitamin as a routine measure. Impending coma is the only situation in relation to hepatitis in which either a specific diet, drug or antibiotic is of value.

#### THE CONVALESCENCE

What are useful guide posts in following a patient with known viral hepatitis? In children, the disease is almost always so transient and mild, that the child can be allowed to set his own rate of mobilization. In an adult, duration and severity of viral hepatitis is quite unpredictable, although the mortality rate is exceedingly low, except in pregnant women and individuals with pre-existing liver damage. In general, therefore, the prognosis for ultimate recovery is excellent. The height to which the bilirubin or transaminase levels rise is not an indication of the severity of illness. However, a patient who continues to be anorectic and whose transaminase persists at a level of a thousand units or more for periods in excess of ten days or so is probably headed for a relatively prolonged course. Needless to say, there are exceptions to this also. Generally, once the transaminase level begins to fall it usually returns to the range of 200 units or less within a week or ten days. At about the same time, jaundice will disappear slowly. The liver will become less tender although it may stay enlarged. The flocculation tests reach a peak at about this time. Once the transaminase has fallen below 200 units and the jaundice has disappeared, bromosulfalein excretion becomes the most reliable index of the patient's recovery, and this determination can be used to indicate when the patient should be mobilized and whether his rate of mobilization is too rapid or not. In general, mobilization of a patient as long as his BSP retention is above 5%, and the level is still falling, will be followed by a relapse in a relatively high proportion of patients.

Our own policy is as follows: As soon as a patient no longer clinically appears jaundiced, even though his serum bilirubin may still be elevated, BSP tests are done on a weekly basis. When they have fallen below 5% retention, the patient is gradually mobilized. He is

followed at intervals of 3-4 days with repeat BSP tests and with daily palpation of the liver. If this organ becomes increasingly tender or a rise in the BSP level is noted, the patient is returned to bed-rest until the dye excretion has again returned to previous levels. As far as palpation of the liver is concerned, it should be recalled that in many women toward the end of a menstrual cycle, the liver will enlarge and become increasingly tender, in the absence of any increase in inflammation. This is due to fluid and salt retention secondary to the normal cyclic change in estrogen level.

If everything goes well, mobilization is continued until the patient has returned to reasonably normal activity. The BSP at this time is a relatively good indication of residual liver damage. If it is at a level of zero to 3%, it is very unlikely that the individual will have further trouble. Flocculation or turbidity tests are of no value as an index of when to begin mobilization or as an assessment of residual liver damage.

#### THE POST-HEPATITIS SYNDROME

Many adults who have had hepatitis will continue to complain for periods of up to a year or more of a dragging sensation in the area of the right upper quadrant and will have occasional transient enlargement of the liver, particularly associated with relatively mild viral infections and various infectious gastroenteritides, which, particularly in women, may be associated with transient period of marked anorexia. Such complaints do not necessarily indicate that the patient has chronic or prolonged hepatitis. In fact, what he has is the post-hepatitis syndrome. Characteristically, these individuals have a history of hepatitis followed by complete recovery but then on repeated occasions develop anorexia, irritability, lethargy, weakness, headaches and right upper quadrant discomfort. Women frequently show menstrual disturbances of various types. Sometimes the liver is enlarged and tender. In very few of these patients are jaundice or abnormal hepatic function tests present. The difficulty of correlating these marked subjective signs with objective evidence of hepatic disease by biopsy or in the laboratory has been recognized. As I indicated earlier, the post-hepatitis syndrome occurs more commonly and more severely in women, and may be almost incapacitating for them. While there is a tendency to write these complaints off as being due to crotchyness it may be stated with certainty that such an attitude is unjustified. The basic problem is that the women's estrogen metabolism has been upset. Detoxification and excretion of the various sex hormones are accomplished to a large extent in the liver. During the following hepatic disease, these functions become deranged, and women, who depend far more on these hormones for their general well being than men suffer more as a consequence. In general, the symptoms of the post-hepatitis syndrome are due to low estrogen levels. Excellent results can usually be obtained by treating the patients with cyclically administered oral estro-



gen; to give them estrogen for about 18 days, to add a progestational agent during the last 4 or 5 days of the cycle and then to abruptly withdraw both hormones. Menstrual bleeding will usually start within a day or two following withdrawal of the drugs. After permitting the woman to bleed for about 7 days, estrogen administration is again started. Unfortunately, this form of therapy is expensive since best results are obtained with the natural hormones; stilbesterol, while more economical to use, is also much less effective. Following the administration of these agents for 2 or 3 cycles, they can usually be withdrawn and the patient's well being will persist. The male suffering from the post-hepatitis syndrome is much more difficult to treat. Androgen administration has not been of benefit and should not be used. Similarly, while it may be tempting to employ tranquilizers to calm down an irritable patient, these have an erratic action with frequent aggravation of symptoms. The best approach consists of reassuring the patient that he will feel well within a month or two, and to see him at intervals, primarily for his own reassurance that nothing is fundamentally wrong.

The question is often raised as to what the incidence of permanent residual following hepatitis is. Unquestionably, no adult with this disease escapes without some permanent damage. On the other hand, chronic progressive liver disease ending with cirrhosis, portal hypertension, and death appears to be exceedingly rare except in people with pre-existing liver damage, or in patients who because of habit or employment continue to be exposed to potent liver toxins, such as alcohol, petroleum products, and other hydrocarbons of a similar nature.

#### ATYPICAL HEPATITIS

In the past several years we have seen outbreaks of illnesses resembling classic hepatitis in their clinical and laboratory manifestations but which must have been due to agents other than the two associated with infectious and serum hepatitis. For example, we saw one epidemic among student nurses in a hospital in the Midwest where an incubation period of approximately 7 through 9 days could be clearly delineated and where, while liver involvement and even icterus were present in most patients, the illness was so mild and transient to be strikingly different from the classic form of hepatitis. For example, the patients with jaundice did not show any appreciable weight loss, although in the usual case, an adult loses at least 10 to 15 pounds. In addition to that, all affected individuals recovered within 5 to 6, in some cases 10 days following the onset of their illness. The short incubation period, as well as the consistently short and benign course of the clinical illness, effectively rule out the diagnosis of either infectious or serum hepatitis in the classical sense even though the clinical laboratory findings did not differ from those usually observed with the latter two diseases. During the course of any year, we see in our clinic

population a fair number of individuals who demonstrate similar peculiar illnesses for which no clear-cut etiologic explanation is at hand. This particular form of hepatitis appears to be more infectious than the classical one, and will spread within a family unit even though gamma globulin has been given to all contacts as soon as a diagnosis is suspected. These observations indicate that viruses other than the two usually considered are capable of producing primary liver inflammation in adults, a fact which has been recognized for several years to apply in congenital hepatitis.

#### CONGENITAL HEPATITIS

Congenital hepatitis is occurring or is being recognized with increasing frequency in all areas of the United States, and this subject therefore is worthy of some discussion. It is now known that this is not a disease but rather a syndrome due to multiple etiologies. At least four viral diseases that may be congenitally transmitted affect the liver of the newborn infant sufficiently to give rise to the signs and symptoms of neonatal hepatitis. These illnesses are cytomegalic inclusion disease, generalized Herpes simplex infection, Coxsackie B virus and serum hepatitis. Coxsackie and herpes infections are relatively rare and usually produce more generalized disease with involvement of many organs and tissues, although the effect on the liver by the latter virus appears to be especially marked.

There is no evidence that the agent causing epidemic or infectious hepatitis causes clinical illness in the fetus or congenital disease in the newborn. Many women who developed hepatitis during their pregnancy have been carefully observed and none of their infants were adversely affected nor was there an increase in the incidence of congenital anomalies. On the other hand, the ability of the serum hepatitis virus to cross the placental barrier and to cause disease in the newborn is well established. Characteristically, the majority of mothers of infants with this congenital disease fail to show signs and symptoms of liver involvement and thus represent asymptomatic carriers of hepatitis B virus. It is unfortunately true that these women generally tend to infect several of their off-spring successively although an occasional one may escape unharmed.

The clinical picture of hepatitis in the neonatal period is that of an obstructive jaundice with whitish or grayish stools and bile but no urobilinogen in the urine. The liver is usually enlarged, the spleen only occasionally. Because of the marked clinical picture of obstructive jaundice, the diagnosis of biliary atresia or other forms of congenital biliary obstruction is usually considered. Unfortunately, laboratory tests are of little value in differentiating obstructive from inflammatory disease in infants; flocculation tests, alkaline phosphatase and the serum enzyme determinations give unpredictable results in this age group. Only a liver biopsy performed under local anesthesia will provide the answer, unless the patient spontaneously improves,

which, of course, rules out atresia. It should be emphasized that infants with liver disease tolerate general anesthesia exceedingly poorly and the mortality rate following its administration is exceedingly high. Infants with congenital hepatitis not due to the herpes virus but to the agents that have been mentioned usually recover. A variable percentage, estimated as high as 25%, develop cirrhosis in early childhood and succumb from this, but the majority appear to do well. Because of this high rate of recovery from hepatitis and the high mortality following surgical intervention, it is particularly important not to subject these patients to surgery in an ill-advised attempt to correct a non-existent biliary atresia.

#### GAMMA GLOBULIN PROPHYLAXIS

While there is no specific therapy for hepatitis, an exceedingly effective method of prophylaxis exists. This is, of course, immune globulin, also called gamma globulin, about which there now is a great deal of valuable information on which concrete recommendations can be based. As far as liver disease is concerned, this biologic is effective only against the virus causing infectious hepatitis; there is little evidence to suggest that the material prevents serum hepatitis. The recommended dose of immune globulin in the prophylaxis of infectious hepatitis is 0.01 ml. per pound of body weight. For practical purposes one usually gives 1 ml. to children under 100 pounds and 2 ml. to older children and adults. This dose is adequate to prevent the occurrence of hepatitis in approximately 90 to 95% of persons to whom it is administered prior to the onset of symptoms. Almost all failures that do occur are due to the fact that the injection was given too late in the incubation period, namely, just prior to the onset of symptoms. To be effective, it is essential that the material be administered at least 5 to 7 days prior to the onset of illness. Since the incubation period of infectious hepatitis is approximately 3 weeks, this does allow the physician ample time for proper diagnosis of the index case before the material need be administered to family contacts. There have been reports in the literature suggesting that the dosage here recommended is too low, these reports are based on abnormal conditions and deal with a hepatitis virus which differs in some ways from the agent encountered in open communities. All data collected in naturally occurring outbreaks indicate that the 0.01 ml. per pound dose is adequate, and that nothing is gained by giving more.

It is important to note that immune globulin never contains the virus of serum hepatitis, since the chemical manipulation of plasma necessary for globulin precipitation either destroys or precipitates the serum hepatitis virus in a different fraction. As a matter of fact, any reactions to gamma globulin administration other than pain are rare if the material is injected by the only possible route, namely intramuscularly. Immune globulin must never be given intravenously, even

in dilute form. Severe, and sometimes fatal reactions have occurred following this type of administration.

#### RECENT RESEARCH ON THE HEPATITIS VIRUSES

In conclusion, I would like to discuss with you some of the recent work aimed at isolating in a laboratory system the agents responsible for human hepatitis. Ever since it was shown at about the time of the second World War that human hepatitis was due to several viruses, the search has been on to develop a laboratory method for the propagation of these agents. Initially, animals were used, and this work was completely unsuccessful. It was found that many mammalian and avian species carry hepatitis viruses of their own, which usually proved species specific. In other words, the virus that produces hepatitis in the dog is unable to produce a similar disease in man, and vice versa. Then, when tissue culture methods became available, attention was focused on attempting to grow the viruses on cells in test tubes. This has proven to be a singularly difficult, disappointing and confusing task. The first report of a possibly successful propagation of the infectious hepatitis virus described the agent's growth in chick egg embryos, and the authors used the fluid from the egg as a skin test antigen, to determine immunity to this virus. Material taken from the infected eggs did in fact on occasion produce a transient clinically evident and laboratory demonstrable form of hepatitis in human volunteers, but this action was far from constant. This virus eventually died out and could not be propagated further. Subsequent reports of the isolation of the infectious hepatitis agent in tissue culture and/or chick egg embryos could not be confirmed. In 1956 a group reported the successful cultivation of cytopathogenic agents from patients with clinical hepatitis in a special cell line known as Detroit-6. The agents thought to produce this effect were rather unique in many respects. The culture system as originally defined required elaborate precautions, and perhaps due to this there were a number of reports of unsuccessful attempts to reproduce the results. The tissue culture was considerably refined in succeeding years and recently the same group presented a comprehensive report covering studies carried out in the virology laboratory and a number of clinic trials in which a group of volunteers received these agents. The results can be briefly summarized. The administration of the tissue culture fluids to the human volunteers produced reasonably consistently a mild illness occasionally accompanied by low-grade fever and by jaundice, as well as changes in the liver chemistries resembling those found with the naturally occurring disease. There are, however, several features which are disturbing. In the first place, the virus in question were isolated originally only from the serum of the initial patients. This in itself is not too unusual since the virus of infectious hepatitis does produce a viremia. However, when the tissue culture

*Continued on Page 74*





DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### State Medical Advisory Committee Activated

GEORGE E. SULLIVAN, M.D., Chairman

#### FOREWORD

After the enactment of the Kerr-Mills legislation in Maine, it became increasingly apparent that there were many problems pertaining to hospital and medical care which needed attention by a representative group of professional persons in an advisory capacity to the Department of Health and Welfare. To focus attention on how to obtain needed and appropriate care for people; not only as related to Medical Assistance for the Aged, but also all categories of public assistance was the objective in our decision to form a Medical Advisory Committee. Much thought was given to the selection of such a committee within the Department and the Maine Medical Association. Those invited to serve represent various medical and paramedical professions and are strongly identified in professional relations among their colleagues. A definite attempt was made to select the persons geographically. It is indeed most gratifying that a committee of outstanding calibre was marshalled. That these extremely busy persons have paid serious attention to the monthly meetings held in Augusta and are delving into a variety of problem areas far beyond what we had envisioned at the outset shows both the nature of the needs and willingness to serve. As you will see by the following report of George E. Sullivan, M.D., Chairman of the Committee, the impetus provided by problems related to Medical Assistance for the Aged has launched the Committee into a far broader field out of which will come, hopefully, greater knowledge and better planning for the medical care of needy Maine citizens. — Dean Fisher, M.D., Commissioner.

The charge to the Medical Advisory Committee for the Department of Health and Welfare was aptly given at its first meeting last July by Dean Fisher, M.D. He stated that the current medical care program is limited to hospital and nursing home care because of lack of available funds to date. But it is vitally important to define the components of an adequate medical care program so that priorities according to medical needs may be established as funds become available. He indicated that the Department has no formulated plans or refined ideas about such a program and is, thus, seeking the advice of this Committee.

The categories of assistance groups for which the state has varying responsibilities include Old Age Assistance (OAA); Aid to Families with Dependent Children (AFDC); Aid to the Blind (AB); Aid to the Disabled (AD); Medical Assistance for the Aged (MAA); State Hospital Aid for medically indigent, and care for specific groups such as committed children.

The Advisory Committee adopted as its statement of purpose the following: "To assist the Department of Health and Welfare in program planning for medical services, i.e.

"A. Establishing priorities in terms of budget limitations and primary basic medical care needs.

"B. Policy formation regarding amounts and types of care with proper quality and quantity controls so

that the Bureau is purchasing at all times the highest standards of medical care attainable in quality as well as in accordance with appropriate medical need and budgeting limitations in quantity.

"C. Continuous liaison function between the Bureau and the professional groups represented for interpretation of the Bureau's program, assistance in resolving problems, and carrying out policy between the Bureau and the professions to enable and maintain the professional cooperation and relationship indispensable to the adequate administration of a medical care program."

Stephen P. Simonds, director of the Bureau of Social Welfare, reported that total expenditures last year for all public assistance categories of medical care amounted to some \$6,134,722, including nursing home care expenditures of \$2,854,000. Estimated expenditure for nursing home care in fiscal 1964 is \$3,250,000. He also affirmed the need for this Advisory Committee for evaluation and planning at the policy level of the Bureau. It is easy to see from the size of these figures the complexities and weighty considerations to which this Committee is exposed. It was pointed out that in many state medical care programs there is provision for other components of medical care in addition to hospital and nursing home care, which are now the only services available for Maine's recipients of public assistance. Doctor Fisher said that since currently there are limita-

tions in state funds for expansion of medical care programs it may be that the six million per year being spent for hospital and nursing home care might be reevaluated to permit other types of care, with the program growing step by step on a priority medical need and budgetary basis. It is hopefully expected that by so doing a more realistic per diem payment for services might be forthcoming. It became obvious during discussions that there might be over-utilization of these services by too lengthy stays and it was shown that cutting one unnecessary hospital day per person would allow about \$300,000 for use in some other area of this program.

The initial meeting of this Advisory Committee revolved around a discussion of length of stays in both hospitals and nursing homes, and the relationships between the two in terms of continuity of patient care, and transfer of patients from hospital to nursing home.

At a subsequent meeting of the Advisory Committee in September the discussion zeroed in on a full-scale analysis of nursing home problems including requirements for licensing, lengths of patient stays, range of payments, standards of care, physicians' services and other related aspects. All available data was presented, both from Department reports and in reference to a 1962-63 Bingham Fund survey of medical care facilities in Maine.

The chairman and members of the Committee agreed that consideration of nursing home care was one of the important interests of this Committee.

After lengthy discussion as to ways of analyzing this problem, it was agreed that more precise knowledge of patient needs was indicated. Since the Department, this Advisory Committee, and the Maine Hospital Association are all interested and concerned about nursing home care, it was voted that a subcommittee of five be appointed to devise ways and means of carrying out a study of the medical, social and rehabilitative needs of public assistance patients in nursing homes. Doctor Fisher suggested that a project of this kind might be assisted with federal money.

Doctor James H. Bonney was named as chairman

and other members of the nursing home study subcommittee are Doctor Carl E. Richards, Doctor Edward T. Newell, Doctor Warren G. Strout and Mr. Wilfred A. Poirier, Executive Director of the Maine Hospital Association.

A report of this subcommittee's work to date will be presented in another issue of the Journal. For the record now, however, may we state that there will be contacts with many physicians and with nursing home administrators from this subcommittee and its teams of study leaders in the various Maine areas. We hope that all who are approached will offer their complete cooperation.

Subsequently another subcommittee of equal importance was named to examine the feasibility of carrying out a pilot project in the Rockland area in cooperation with the county medical societies to test the meaning and cost of coordinated home care services for the state's recipients of public assistance. Doctor Ernest W. Stein was named project chairman and a report of his committee's decisions and work will be given in more detail in a later issue of the Journal.

As chairman of the Medical Advisory Committee I wish to express my appreciation for the attendance and interest shown by members as well as the helpfulness of the staff of the Department of Health and Welfare. With our united efforts it would seem that much can be accomplished in this new and important endeavor.

#### MEDICAL ADVISORY COMMITTEE MEMBERS

Merle S. Bacastow, M.D., Matthew I. Barron, Roswell Bates, D.O., Robinson L. Bidwell, M.D., James H. Bonney, M.D., George W. Bostwick, M.D., Milan A. Chapin, M.D., Dexter J. Clough, II, M.D., Llewellyn W. Cooper, M.D., Carleton F. Davids, Philip S. Fogg, Jr., M.D., Richard J. Goduti, M.D., Elmo G. Hall, Daniel F. Hanley, M.D., Charles A. Hannigan, M.D., Harry M. Helfrich, Jr., M.D., James L. Hood, John J. Lorentz, M.D., Charles D. McEvoy, Jr., M.D., Thomas A. Martin, M.D., Edward T. Newell, D.O., Everett A. Orbeton, M.D., Douglass C. Penoyer, M.D., Wilfred A. Poirier, Loring W. Pratt, M.D., Philip Reiman, Carl E. Richards, M.D., John A. Root, M.D., Albert P. Royal, Jr., M.D., John W. Smith, Jr., Ernest W. Stein, M.D., Warren G. Strout, M.D., George E. Sullivan, M.D., William F. Taylor, M.D., Philip P. Thompson, Jr., M.D., Raymond Walton, R.N.





*These are the **DAYS***

*. . . to mark on your calendar*

<b>JUNE 1964</b>						
S	M	T	W	T	F	S
	1	2	3	4	5	6
7	8	9	10	11	12	13
<b>14</b>	<b>15</b>	<b>16</b>	17	18	19	20
21	22	23	24	25	26	27
28	29	30				

The 111th Annual Session

of the

Maine Medical Association

at

The Samoset, Rockland, Maine

## *The Program*

*for this Annual Session includes . . . .*

### SCIENTIFIC SESSION SPEAKERS

- Huggins of Boston, Massachusetts on "Frozen Blood"
- Hungate of Kansas City, Missouri on "Medical Management and The Emergency Treatment of Disaster Victims"
- Dunlop of Worcester, Massachusetts on "Hypovolemic Shock"
- Kinsell of Oakland, California on "Hypertension"
- Fager of Boston, Massachusetts on "Pituitary Oblation in the Treatment of Diabetic Retinopathy"
- Adelson of Cleveland, Ohio on "Forensic Medicine"

### AFTER DINNER SPEAKERS

Internationally famous after dinner speaker, DR. R. C. S. YOUNG of Birmingham, Michigan and

DR. JOHN C. KRANTZ, JR. of Baltimore, Maryland, author of the book "A Portrait of Medical History and Current Medical Problems"

*Watch your mail for . . . .*

PROGRAM DETAILS

SPECIALTY GROUP PROGRAMS

PROGRAM FOR THE LADIES

RESERVATION FORMS



## *The President's Page*

October 31, 1963

Ernest W. Stein, M.D., President, M.M.A.

Dear Doctor Stein:

. . . I am writing this letter primarily to set forth essentially the remarks which I made yesterday in relationship to the billing which physicians may make to the patients for whom the Department of Health and Welfare has some responsibility of varying degree.

1. For a child who has been committed to our custody by the court, and who is what is commonly referred to as a "State child," we pay physicians' fees for any necessary or properly authorized services. These charges will be authorized by the foster parent under emergency conditions or for minor services, or by the responsible child welfare worker when the necessary services are extensive in kind or are not emergency in nature. Normally, charges for these services are made on the basis of the physician's usual scale. If our worker feels that there is any necessity for negotiation in a given instance, we usually tend to use the Blue Shield scale as a guide. There will be some instances where a committed child will be admitted to a hospital as a ward patient, and under these circumstances the physician may not be permitted to charge for his services by his own hospital or staff by-laws. This, however, is not our decision nor our responsibility.
2. In all four public assistance categories — that is, old age assistance, aid to the blind, aid to dependent children, and aid to the disabled, we do not enter in any way into the question of physicians' fees. This is purely a matter to be arranged between the physician and the patient on whatever terms are mutually agreeable. We are not involved and not concerned in any arrangement that they may make. Obviously, the public assistance recipient is in highly straitened financial circumstances, but whether the patient is billed or not and the basis for such billing is not of our concern.
3. We do not enter in any way into the matter of billing, charges or fees in relation to medical assistance for the aged beneficiaries. This, again, is a matter which is purely between the physician and the patient and they are perfectly free to arrive at any agreement they may choose.
4. The hospital aid patient who is medically indigent, under age sixty-five, and not included in the public assistance program, is, again, an individual for whom our responsibility is limited to the making of a contribution towards the payment of his hospital bill, and, again, we do not enter into the matter of fees, billing, or charges that may be made by the physician in any way whatsoever.
5. The general relief patient is purely the responsibility of a municipality and the circumstances under which charges may be authorized, the extent of the charges, and similar matters are determined by the local overseer of the poor. General relief is available to an individual only upon application by the individual concerned. The decision to extend or not to extend general relief is made by the local official. In the few instances where the general relief recipient does not have a local settlement, we may finally be in a position of reimbursing the local official for any expenditures he may have made. Under these conditions, we may on some occasions protest the amount of fees charged and may ask for explanation or justification.

In hospital aid, medical assistance for the aged, and public assistance, we assume that the patient will be hospitalized at ward rates for our payment is a flat all-inclusive per diem payment. However, the hospital may admit the patient to any accommodation it chooses as long as neither we nor someone else is expected to pay a differential in cost based upon the placement of a patient in accommodations more expensive than ward accommodations. We are not involved in any way in arranging for the hospital admission of any patients except some instances in child welfare and occasionally a few instances in general relief. Therefore, by and large, we are not involved in selection of physicians, and neither are we involved in the question of whether or not a private patient relationship between a patient and a physician will or will not continue after the patient has been admitted to the hospital. This is purely a matter of hospital and staff by-laws or practices.

Sincerely yours,

DEAN FISHER, M.D., *Commissioner*

## Maine Heart Association Notes



### Prophylaxis Of Rheumatic Fever

"Once a patient has had rheumatic fever, he is stigmatized by the attack as having a susceptibility to streptococcal complications. In such patients, the risk of rheumatic recurrence is too great for prevention by a find-then-treat approach to streptococcal infections. Therefore, prophylaxis against *recurrences* of rheumatic fever consists of the continuous use of antimicrobial agents to prevent streptococcal infections before they can occur.

"Of the regimens available for continuous prophylactic purposes, the most effective has been a monthly injection of benzathine penicillin G, 1,200,000 units. . . . Of the well-tested oral regimens, sulfadiazine, 1.0 Gm. daily, has been as effective as potassium penicillin G, 200,000 units once or twice daily. . . . As prophylaxis against rheumatic recurrences, oral penicillin might be more successful if its continuous low daily dosage were augmented intermittently by therapeutic courses. Such a regimen is now receiving clinical trials.

"The major unresolved contemporary issues in antirheumatic prophylaxis are who should receive it and for how long. It currently seems reasonable to give prophylaxis to all rheumatic patients for the first few years after the acute attack . . . and to maintain prophylaxis in children (who are most exposed to streptococcal infections) until the completion of high school. Thereafter, the decision in regard to prophylaxis is still controversial. Some authorities believe that prophylaxis should be maintained in all rheumatic patients indefinitely, i.e., for life. Others . . . believe that the need for continued prophylaxis should, with our present knowledge, be treated individually, depending on the age and cardiac status of the patient, the risk of rheumatic recurrence, and the risk of cardiac damage in the recurrence. Research now in progress may help define these risks more accurately and provide better data for the clinical judgment.

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Feinstein, Alvan R. American Heart Journal, Volume 67, pages 278-279, 1964.

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### Announcement

Enrollment applications for the Seventh Inter-American Congress of Cardiology, to be held at Montreal, June 14-19, 1964, can be obtained by writing the Maine Heart Association, 116 State Street, Augusta, Me. All scientific meetings and the Commercial Exhibition will be located on the Convention Floor of the Queen Elizabeth Hotel. Immediate enrollment is requested to insure hotel accommodations.



in virtually all diarrheas...prompt symptomatic control

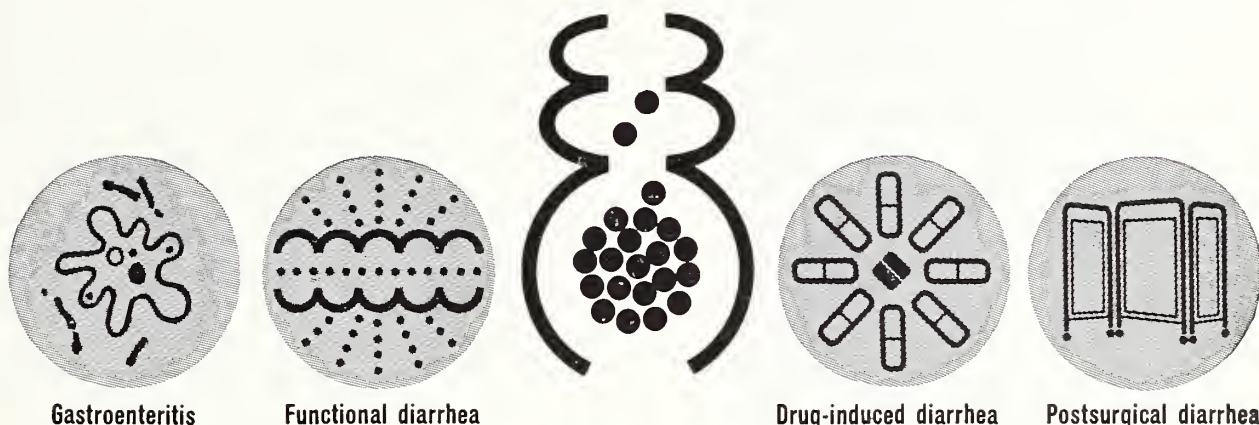
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Lomotil controls the basic physiologic dysfunction in diarrhea—excessive propulsive motility. Pharmacologic evidence indicates that it does so by directly inhibiting propulsive movements of the intestines. This direct, well-localized activity controls diarrheas of widely varied origin and does so promptly, conveniently and economically.

The relatively few conditions in which Lomotil has given less than satisfactory control have been, for the most part, those such as severe ulcerative colitis in which too little anatomic or functional capacity of the intestines remains for the motility-lowering action of Lomotil to have effect.

It should be noted, however, that Lomotil has proved highly useful in mild to moderate ulcerative colitis and in several other refractory forms of diarrhea.

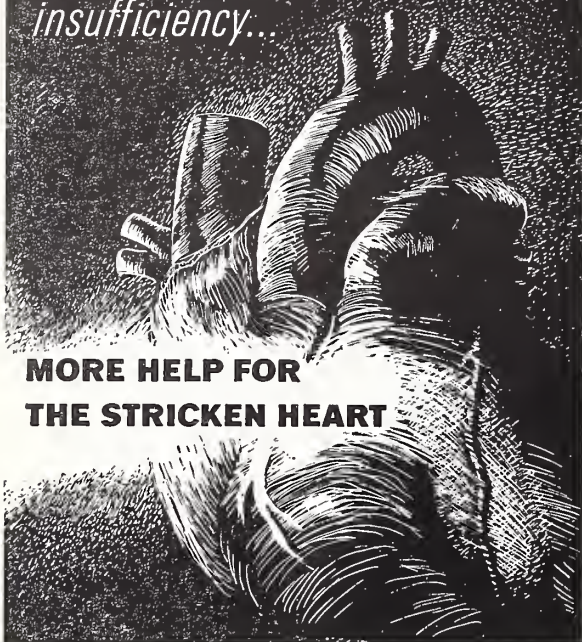
*The recommended initial adult dosage* is two tablets (2.5 mg. each) three or four times daily, reduced to meet the requirements of each patient as soon as the diarrhea is controlled. Maintenance dosage may be as low as two tablets daily. *Children's* daily dosage (in divided doses) varies from 3 mg. for a child of 3 to 6 months to 10 mg. for one 8 to 12 years of age. Lomotil is an exempt narcotic; its abuse liability is low and comparable to that of codeine. Recommended dosages should not be exceeded. Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdose.

*Research in the Service of Medicine*

**SEARLE**

CLINICAL DIAGNOSIS AND MANAGEMENT OF  
HEPATITIS PATIENTS — *Continued from Page 68*

*In long-term  
treatment  
of your patients  
with coronary  
insufficiency...*



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THE STRICKEN HEART**

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material containing the virus was fed to volunteers, no disease occurred, yet it is known that the primary spread of the infectious hepatitis virus is via the alimentary tract. Infection in volunteers could only be produced if the agent was injected parenterally. Then, the incubation period of the experimental disease was longer than that normally seen with infectious hepatitis, averaging consistently more than 30 days. You will recall that the incubation period of the natural epidemic disease is approximately 21 days and at least 45 days for serum hepatitis. Then, the duration of viremia in the volunteer was longer than one would expect to find in patients with infectious hepatitis. Another feature which I find disturbing in the analysis of these data is the fact that the attack rates among the volunteers receiving the material was higher than one would expect. It is well known that the great majority of young adults has had hepatitis in the past and is relatively resistant to infection, whether they in fact have a clinical history of infectious jaundice or not.

What does all this add up to? It believe one can say that this particular group of investigators has in fact isolated several agents related to each other but probably serologically distinct, which are able to produce transient liver inflammation in human beings following parenteral injection. They may be dealing with an atypical serum hepatitis-like virus. There is no evidence that these agents are in fact the one or ones responsible for epidemic infectious hepatitis as we see it in the country at large. This particular point requires a great deal more evidence than is available at the moment. Certainly, there is no justification for the "hoopla" that appeared in the lay press, who generally interpreted these data as indicating that a hepatitis vaccine was just around the corner.

It should be mentioned that in the past two years other investigators have reported the recovery of cytopathogenic agents from patients with infectious hepatitis and from plasma known to produce serum hepatitis, but in these studies the evidence that the viruses are in fact related to the disease in question is even more tenuous than in the investigations that I have cited in detail. We are, therefore, only at the beginning of this work, it will prove extraordinarily difficult to establish clearly the relationship of any agent isolatable by laboratory means as one of the several causes of human hepatitis. Certainly, even talk of a successful vaccine should be postponed until the time that these etiologic relationships are fully understood.

**FOR SALE**

The home and office of the late Silas A. Coffin, M.D. of Bar Harbor, Maine are for sale.

House is partially furnished; office with equipment.

Write to Mrs. Phyllis C. Coffin, 39 High Street, Bar Harbor, Maine.



## Necrology

LUTHER A. BROWN, M.D.

1875-1964

Luther A. Brown, M.D., 88, of Portland, Maine died on February 22, 1964. He was born in Yarmouth, Nova Scotia on July 11, 1875, son of Benjamin and Maria J. McKinnon Brown.

Dr. Brown attended public schools in Lynn, Massachusetts, was graduated from Tufts College and received his medical degree from Boston University Medical School in 1901. He began the practice of medicine in Portland, Maine in 1902 following an internship at the Trull Hospital in Biddeford, Maine.

Dr. Brown was an Honorary member of the Maine Medical Association and the Cumberland County Medical Society, having received a 50-year pin in 1951, a 55-year pin in 1956 and a 60-year pin in 1961. He was also a member of the American Medical Association, a former President of the Cumberland County Medical Society and Portland Medical Club, a member of Immanuel Baptist Church, Portland Lodge, AF & AM, Maine Consistory, Valley of Portland and was a 32nd degree Mason. He was on the staffs of the Maine Medical Center and Mercy Hospital in Portland.

Surviving are a daughter, Ruth E. Brown of Sylmar, California; a grandson, Luther B. Francis of Portland, Maine and a granddaughter, Mrs. James Ferris of Canton, Pennsylvania.

## County Society Notes

### KENNEBEC

The Kennebec County Medical Association held its monthly meeting on January 16, 1964 at the Senator Motel in Augusta, Maine.

The President, Kenneth W. Sewall, M.D., conducted a brief business meeting following dinner.

Paul H. Pfeiffer, M.D. reported on the recent meeting of the Council of the Maine Medical Association in which he discussed the financial status of the Journal and the present functioning of the Maine Medical Education Foundation as a part of the recruitment program here in Maine.

George J. Robertson, M.D., at present associated with the Pratt Clinic - New England Center Hospital and the Bingham Associates Fund, introduced the speaker of the evening, Robert P. McCombs, M.D., Chief of the Allergy and Pulmonary Service at the Pratt Clinic and Director of Postgraduate Teaching at Tufts University Medical School. Dr. McCombs discussed Sarcoidosis and Pulmonary Insufficiency by presenting a case and asking for discussion of the case in the manner of a clinical pathological conference. This was a thought provoking and stimulating method of presentation and led to wide discussion.

EARLE M. DAVIS, M.D.  
*Secretary*

### PENOBSCOT

The Penobscot County Medical Society met on January 21, 1964 at The Tarratine Club in Bangor, Maine.

Ernest W. Stein, M.D., President of the Maine Medical Association, discussed the reappointment of Dean H. Fisher, M.D. as Commissioner of Health.

Mr. George Nilson, Field Director of the Bingham Associates Fund, discussed the history of the Fund as well as its present role.

George J. Robertson, M.D., Medical Director of Bingham

Associates, discussed the contemplated program of the Bingham Associates in the postgraduate medical educational area in Maine. Specifically he described the educational TV programs which are to start March 3, 1964 at 10:20 p.m., the resident and teaching fellow exchange program starting in the Kennebec Valley Hospital area and the availability of short term resident appointment programs for postgraduate education available to physicians from Maine.

HADLEY PARROT, M.D.  
*Secretary*

### HANCOCK

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on February 12, 1964.

William W. Ward, M.D. of Rockland, guest speaker, presented a well illustrated talk on Diagnosis and Treatment of Abdominal Surgical Emergencies in the Newborn. A lively question and discussion period followed.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

### LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on February 18, 1964. Fifteen members were present.

The film, Kerr-Mills Medical Assistance for the Aged, was shown and Nelson P. Blackburn, M.D. of Bath, presented an interesting C.P.C.

GEORGE W. BOSTWICK, M.D.  
*Secretary*

### SOMERSET

The Somerset County Medical Society met at the Village Candle Light in Skowhegan, Maine on February 18, 1964.

The President, W. Edward Jordan, Jr., M.D., presided at the business meeting.

Robert A. Bearor, M.D. of Portland, the guest speaker, spoke on Practical Aspects of Radiology in Medicine. A question and answer period followed.

MARIAN L. STRICKLAND, M.D.  
*Secretary*

### ANDROSCOGGIN

A meeting of the Androscoggin County Medical Association was held at the Central Maine General Hospital in Lewiston, Maine on February 20, 1964. Henry C. Thacher, M.D., Vice-President, presided in the absence of the President, Robert D. Wakefield, M.D.

The guest speaker, Peter W. Bowman, M.D., Superintendent of the Pineland Hospital and Training Center, was introduced by Paul M. Beegel, M.D. Dr. Bowman spoke on The Dynamic Approach to Mental Retardation. A question and answer period followed.

John W. Carrier, M.D. reported for Operation Hometown and Charles A. Hannigan, M.D. reported on the various studies being performed by the Health and Welfare Department.

It was voted that (1) The Androscoggin County Medical Association endorse Visiting Nurse Service for Androscoggin County; (2) A subcommittee be appointed by the chair to act in behalf of the society for obtaining visiting nurse services through whatever means and facilities are or will be available, also to keep the society informed of the progress of this plan.

DONALD L. ANDERSON, M.D.  
*Secretary*

**New Members****AROOSTOOK**

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 Presque Isle

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William C. Bromley, M.D., Medical Ctr., Herrick Rd., South-  
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 Joseph H. LaCasce, M.D., 50 Union St., Ellsworth  
 Earle W. Spencer, M.D., Maine Coast Mem. Hosp., Ellsworth

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Price A. Kirkpatrick, M.D., Thayer Hospital, Waterville

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# The Journal of the Maine Medical Association

Volume Fifty-Five

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No. 5

## Time Patterns in Headache Their Diagnostic and Therapeutic Significance

E. CHARLES KUNKLE, M.D.

When recurrent headache is a diagnostic problem elaborate studies are sometimes undertaken; the best and most immediate clues, however, are often found in a full history. Of particular value are data concerning the time patterns of the headache; neither the location nor the intensity of the pain is as useful a guide to the source of the illness. This report offers an analysis of certain significant features in the timing of common headaches, notably those of migraine.

### "RELAXATION" HEADACHE

Among the best documented patterns of migraine is the tendency of the attacks in some patients to occur with "change of pace," during periods of relative relaxation after sustained activity.<sup>1</sup> Headache may occur quite regularly each Saturday morning or afternoon or, especially noteworthy, after sleeping late on Sunday morning ("weekend" headache). It may also occur almost predictably on the first day of a planned vacation. This tempo is perplexing to the patient and may lead him to doubt any interpretations by his physician that the illness is related to life stress. Yet, from the evidence offered by many perceptive patients, it seems quite clear that abrupt "slowing down," particularly after intensive effort, whether this be creative or competitive in the business world, is for many individuals a difficult matter. To the human animal the change to a time of relaxation signifies non-productiveness and lack of outlet for inner drives. On the other hand, some patients will concede, in candid moments, that a weekend at home is actually far from relaxing, perhaps because of the pressures exerted by demanding children and a

difficult wife. Faced with such a situation, the husband may actually feel less harassed and more content in the protective surroundings of his office during the work week.<sup>2</sup>

Attention focused on these aspects of the illness is often the most direct road to improvement. Even though tension-producing problems may resist prompt solutions, the patient's attitudes toward them can often be modified in a favorable way. In addition, he should attempt to shift some of his weekday duties to the weekend, to diminish abrupt changes in routine, and should avoid sleeping late on weekend mornings, if this habit has been likely to induce headache.

### NOCTURNAL HEADACHE

In some individuals migraine or, much less commonly, muscle-contraction headache begins during the night, awakening the patient from sleep.<sup>3</sup> Such timing, like that of "relaxation" headache, seems to the patient quite inconsistent with any thesis linking his disorder to emotional tensions. Accordingly, the physician must be prepared to offer some reasonable interpretations of this phenomenon. The full story is as yet quite incomplete, but at least two hypotheses can be offered: In sleep the central mechanisms for cranial vasomotor control (in part within the brain stem but also at higher levels) may work less efficiently, permitting some relaxation of arterial tone; this then may predispose somehow to the augmented but usually localized vasodilatation which is the major part of a migraine attack. On the other hand, evidence from various sources, notably the content of dreams, suggests that deep-seated con-

flicts are far from dormant in sleep and can readily give rise to trouble, even in the form of a migraine headache. Regardless of the full explanation, headaches recurring mainly or entirely during the night may often be helped by preventive medication. Provided the headache is migrainous and of the type responsive to ergotamine, this drug in one of its various forms can be taken before retiring. Perhaps most effective for this purpose is the rectal suppository containing ergotamine and caffeine (Cafergot®). Regular bedtime use of ergotamine for prophylaxis is worth a trial when nocturnal headache is occurring with high frequency, almost nightly, and over relatively short periods, as so often occurs with "cluster" headache (discussed in a following section). Nightly administration of this drug for a prolonged period, however, is not only costly but potentially dangerous; it may lead to excessive constriction of a coronary or limb artery. If ergotamine is ineffective or if therapy must be long continued, a vasoconstrictor of different type (and less potency) such as amphetamine or dextro-amphetamine, in 5 to 10 mg. amounts by mouth, can be tried, although the value of these agents used in this way has not yet been fully confirmed.

#### MENSTRUAL HEADACHE

In one way or another the menstrual cycle is a matter of concern to most human females, and at times also to their men. Some women prone to vascular headache report that the attacks are closely related to this biologic rhythm, the headache commonly occurring at the onset of the menses or shortly before.<sup>4</sup> In some of these instances, however, a close analysis of the actual tempo of the attacks will indicate that, despite what the patient may have assumed, the headaches occur quite erratically, with no dependable relationship to the menstrual cycle.

In the few women whose headaches are fairly consistently either premenstrual or early menstrual, the physiologic basis for this timing is still unclear. One hypothesis, that fluid retention somehow leads to stretch or local edema of cranial arterial walls, has not yet been supported by any clear evidence. It is quite possible that the hormonal alterations which underly the menses may also in some individuals decrease arterial tone, either at a central or peripheral level, but this, too, is a loose conjecture. Of greater importance than fluid balance or hormonal vasomotor factors may be the complex mood changes which so often foreshadow and accompany the menstrual period. Some women in this phase are notably susceptible to feelings of frustration, resentment, and other adverse reactions — potent sources of headache.

In actual practice, attempts to prevent menstrual headache by the administration of diuretics, such as chlorothiazide or ammonium chloride, during the week before the expected menses usually prove fruitless. The failure probably has many explanations, including

limited pharmacologic activity of the medications used and the inevitable difficulty in accurate timing of the therapy due to irregularities in the menstrual cycle.

#### CLUSTER HEADACHE

The most striking and in many ways the most intriguing tempo of headache is that encountered in patients with the variety of migraine which is now commonly described as cluster headache.<sup>5</sup> This is the headache which is restricted largely to males, is usually localized behind and about one eye, is of high intensity but fortunately brief duration (usually less than two hours and sometimes less than 20 minutes), and is rarely accompanied by nausea or vomiting. More than 9 out of 10 patients with this disorder present a series of headaches in close sequence, usually once each 24 hours and sometimes more, and mainly during the night. Such a cluster of attacks commonly lasts for several weeks, then stops abruptly or tapers off, to be followed by complete remissions lasting months or even years. This remarkable illness has received various names. In earlier years in England it was termed "migrainous neuralgia," and more recently in this country "histaminic cephalgia" (although there has been no convincing evidence that histamine plays any significant part in the mechanism of the headache).

In many instances the explanation for the curious tempo of the attacks is completely hidden, even to close scrutiny, including a search for seasonal or other long-term changes in eating patterns, work load or domestic stresses. In some patients, however, impressive evidence has been found to indicate that the advent of a cluster follows some special and difficult problem in life adjustment. There are a few patients, moreover, in whom a precipitating factor seems to lie in a subtle change in mood, perhaps of endogenous origin preceding the onset of a cluster by several days or weeks.

Recognition of the phenomenon of clustering is crucial in planning drug and other therapy, and in any attempt at evaluation of a treatment program. Failure to recognize the natural history of this disorder, particularly the built-in rhythm which will usually terminate a cluster within a few weeks regardless of what is done, has led in the past to erroneous inferences about the value of certain remedies. This has been notably true of the conclusions about the efficacy of "desensitization" to histamine, a program which now appears to be of dubious, if any, value. Some patients with cluster headache can be helped by ergotamine, so timed as to block the attacks. This is most feasible when the headaches are largely or entirely nocturnal; the drug, most conveniently used in the form of the Cafergot® rectal suppository, can then be given nightly at bedtime, and the treatment withheld every 5th or 6th night to determine when the cluster has reached its natural end. An alternative plan, the use of methysergide (Sansert®), starting with a 2 mg. tablet three or four times a day,

*Continued on Page 82*



# Double Gallbladder

ALFRED HURWITZ, M.D.\*

According to Gross,<sup>1</sup> the term "double gallbladder" is used to describe 28 examples of duplication of the vesica fellea in each of which there were two separate bladder cavities and two separate cystic ducts. The accessory gallbladder may be situated in close proximity to the normal one or along the gastrohepatic ligament. The accessory gallbladder may be larger or smaller than the normal one or the same size.

Duplicate gallbladder may appear as two rows of positive-shadow gallstones or as two distinct shadows in the cholecystogram. When the accessory gallbladder is inflamed or contains calculi, the symptoms are indistinguishable from those occurring with cholecystitis or cholelithiasis in a normally formed gallbladder.

The following case report emphasizes the difficulty in making the diagnosis of accessory gallbladder and the existence of an unusually situated cystic duct.

## CASE REPORT

H. G., a 57-year-old white female entered Maimonides Hospital on April 20, 1961, with the chief complaint of upper abdominal pain, yellow sclerae and the passage of dark urine of four days' duration. The patient had had mild attacks of intermittent, upper abdominal pain radiating to the back for approximately 20 years. During the four months prior to admission, the patient had two discrete attacks of RUQ and mid-epigastric colicky pain radiating to both shoulder blades. The attacks lasted two hours and seemed to be relieved by rest. The last attack occurred two weeks before admission and was characterized by the same type of pain together with chilly sensations. Her urine became dark and her sclerae yellow. A cholecystogram was done at that time and revealed a well-filled gallbladder.

The patient was a well-developed, fairly well-nourished female in no acute distress. Blood pressure was 140/70; pulse 80 and regular. Sclerae were icteric. Tenderness was elicited in the RUQ but the abdomen was soft. The liver edge was smooth and non-tender and palpated 2 cm. below the right costal margin.

Laboratory data revealed a yellow, acid urine with a negative test for bile. The hematocrit was 42%; white blood cell count was 4,100 with 52% polymorphonuclear leukocytes, 43 lymphocytes, 2 eosinophiles and 3 monocytes. Total bilirubin was 2.6 mgms per cent and total cholesterol was 240. SGOT was 138 and SGPT was in excess of 125. A gallbladder series was read as revealing a well-visualized gallbladder with no calculi (Fig. 1). An upper gastro-intestinal series was normal.

She was observed in the hospital by her physician for one week during which she was given demerol repeatedly because of the severity of the RUQ pain. Her urine remained dark and sclerae yellow. Laparotomy was advised because of persistence of the pain, elevation of serum bilirubin and normal liver function tests.

On May 3, 1961, two distinct gallbladders were found at operation. After removal of the normal gallbladder which was



FIG. 1. Two distinct gallbladder shadows can be seen. One represents the normal, elongated gallbladder and the other lying just below the twelfth rib is the round, calculous accessory gallbladder.

in the usual position and appeared normal, a smaller accessory gallbladder situated under the right lobe of the liver was visualized. Roentgenographs were taken on the operating table by injecting hypaque into the lumen of the gallbladder and also by filling the accessory gallbladder by injecting the dye into the cystic duct stump of the normal gallbladder and observing its flow into the right hepatic duct and finally into the abnormally situated cystic duct (Figs. 2 and 3). Both gallbladders were excised. The patient had an uneventful convalescence except for the drainage of a moderate amount of bile around the Penrose drain for several days. She was discharged in good health on May 18, 1961.

## PATHOLOGICAL EXAMINATION

The accessory gallbladder measured 2.5 cm. in diameter and 1.5 cm. in length. The external surface was congested and dull. The mucosal surface showed numerous focal hemorrhagic areas and granular, bile-stained foci. This gallbladder contained numerous, gravel-like, black calculi which were cholesterol by chemical test. Microscopically there was diffuse fibrosis of the wall and inflammatory infiltration of the mucosa. Deep Rokitansky-Aschoff sinuses were present. Separately received was a normal gallbladder measuring  $8.5 \times 3.2$  with a 1.5 cm. attached cystic duct. The mucosa was velvety and its microscopic

\*Courtesy Staff, Mercy Hospital; Former Chief of Surgery Maimonides Hospital, Brooklyn, New York and Professor of Surgery, Downstate Medical Center, New York.

1. Gross, R. E.: Congenital Anomalies of the Gallbladder. A Review of 148 Cases with Report of a Double Gallbladder. *Arch. Surg.*, 32:131, 1936.



FIG. 2. After the excision of the normal gallbladder, hypaque was injected into the accessory gallbladder. The junction of its cystic duct with the right hepatic duct can be seen.



Fig. 3. The accessory gallbladder is filled in a retrograde manner by injecting the dye through a ureteral catheter in the cystic duct stump of the normal gallbladder.

appearance was normal. Diagnoses were (1) normal gallbladder and (2) chronic cholecystitis and cholelithiasis in an accessory gallbladder.

DISCUSSION

The diagnosis of double gallbladder should have been made on viewing the cholecystograms preoperatively rather than retrospectively. Operation might have been postponed unnecessarily if the patient had not become jaundiced. The conviction that the patient had a pathological gallbladder dictated the excision of the normal-appearing gallbladder in the belief that it might be harboring small, soft calculi of the cholesterotic variety. After removal of the normal gallbladder, the calculous accessory gallbladder first came into view and its excision was followed by an uneventful postoperative course.

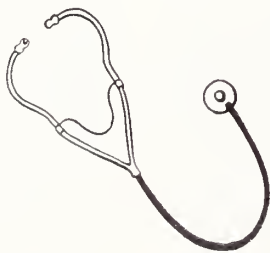
It is suggested that in the rare patient who has experienced attacks of upper abdominal pain suggestive of gallbladder disease, but with a normal cholecystogram,

careful search for an accessory gallbladder be made. In the case presented, failure to find the accessory gallbladder would have resulted in a recurrence of her symptoms. A cholelithogram via a ureteral catheter in the cystic duct of the normal gallbladder revealed the presence of the accessory gallbladder. The advisability of making preoperative preparations to obtain X-ray studies on the operating table is obvious.

CONCLUSIONS

A case of double gallbladder is described. The calculous accessory gallbladder drained into the right hepatic duct via a short cystic duct. Careful search for an accessory gallbladder which includes a cholecystogram or cholelithogram on the table should be made if the gallbladder visualized and palpated at operation appears to be normal.

32 Deering Street, Portland, Maine





# A Health Information Center for the Greater Bangor Area

MIRIAM M. CAMPBELL, M.P.H.

The Bangor-Brewer Health Information Center, operated as a community service by the Bangor-Brewer Tuberculosis and Health Association since 1960, owes its existence to the conviction on the part of the Board of Directors that community resources in the area available to individuals with health problems were not being fully utilized. Although there existed a number of obvious gaps in the network of community health services, it was apparent that the existing facilities and services were not being fully utilized. For various reasons they were unknown or were overlooked by physicians and others seeking care.

It was therefore voted in March of 1960 that the TB and Health Association would establish and operate a Health Information and Referral Service to be known as the Bangor-Brewer Health Information Center.

The objectives were two-fold: (1) to collect and maintain up-to-date information on all available community facilities and agencies offering service to individuals with health problems; (2) to provide referral services to individual patients or others requesting this information.

Although certain gaps in community health services became apparent during the early operation of the Health Information and Referral Service, the primary purpose of the Center was to ensure full utilization of existing services rather than to point up lack of services.

The Center is headed by the Executive Director of the Association who has a masters degree in public health. She is assisted by a staff member who is a registered nurse and by the office secretary.

No intensive publicity campaign was carried out in connection with the offering of this new community service, since it was the opinion of the Board that the information gathering phase should stay well ahead of the demands for service in order to avoid disappointment when answers to questions could not be readily given.

Patients or others seeking information qualify for service without any prior investigative or case-work procedures. Only enough information is obtained from the inquirer to allow determination of the suitability of the service recommended. No attempt is made to determine the specific eligibility of the person involved. This is left to the agency or agencies to which the referral is made.

The service is used by social workers, doctors, nurses, and others trying to arrange suitable care for a client or patient. It is used by individuals trying to obtain help for a family member or for a friend.

Requests for service involve a wide range of problems:

- A young wife whose husband is in the service and hospitalized for TB wants to know where she can have her children checked.
- A grandmother whose grandson was found to have diabetes when he was trying to enlist in the Navy wants help with a diabetic diet.
- A wheelchair is needed by a medically indigent amputee.
- A young husband and father whose wife has been rushed to the hospital inquires about Visiting Homemaker Service.
- A serviceman's wife whose mother is visiting from Germany and is under medical supervision must have regular blood pressure readings.
- A woman is putting off necessary surgery because she and her husband are living on a pension and there is no money for medical or hospital bills.
- A woman living in a trailer development wants to know how she can have their drinking water tested.
- A teen-age boy who saw our sign wants to know where the Good Samaritan Home is located.
- A representative from a church service group wants to know if we know any partially sighted person who would like some sermons done in extra large type.

Although a record is kept of each call, no organized attempt is made to determine whether the referral made was satisfactory.

An analysis of requests for service to date shows that most individuals learned of the service either through the listing in the "yellow pages" under "Health Service" or through the sign on the Tuberculosis and Health Association headquarters.

Although the Health Information Center has been tailored to meet local needs, it is patterned somewhat after the Milwaukee Central Agency for the Chronically Ill which has been in operation since 1948.

In connection with the Center the Tuberculosis and Health Association operates a Hospital Supply Loan Cupboard through which medically indigent patients may obtain hospital beds and other equipment needed for home care.

Also in an attempt to help fill an existing gap in community health services the TB & Health Association has co-sponsored on a demonstration basis the Penobscot Valley Visiting Homemaker Service.

A Tuberculosis and Health Association's program is necessarily concerned primarily with control of respiratory disease including tuberculosis. However, the operation of a central health information service and participation in improving community health services often results in improved care for patients who are the immediate concern of the agency.

In January 1964, the Board voted to extend the Health Information Center scope to provide a clearing house of information on educational materials on smoking and health as well as on the respiratory diseases.

As the Health Information Center begins its fourth year of service to the community, a Directory of the most commonly used resources has been compiled. This

Directory is being distributed to physicians, clergymen, police, nurses, social agencies and others who are concerned with the general area of patient care. The format of the Directory is such that it can be brought up to date periodically with a minimum of expense.

If well received, agencies and individuals on the Directory Mailing List will be provided with revisions annually or as needed.

The Health Information Center and Directory service are meeting a local need and to date are being operated without any increase in Association staff. It is hoped that the Center may provide a model or prototype for other similar health information and referral services in Maine.

#### TIME PATTERNS IN HEADACHE — *Continued from Page 78*

also demands a close watch for the spontaneous remission in the cluster which sooner or later is likely to occur. Accordingly, if the patient does well on this medication, within two weeks the dose of Sansert can gradually be reduced, the ultimate aim being its complete withdrawal, and then its resumption if and when the next cluster begins.<sup>6</sup> Just as important as the choice of a drug is attention to emotional components in the illness. Some form of brief psychotherapy is often warranted.

If the disorder does not readily come under control by such measures as have been described, a useful step is admission of the patient to the hospital for several days of rest and observation. In some way, this procedure will often lead soon to a partial or complete remission in the attacks, regardless of what medication may be administered in the hospital. Such a response may reflect the non-specific effects of a change in life routine and the reassurance offered by thorough in-patient medical study.

#### SUMMARY

In the care of patients with recurrent headache, a complete history is of prime value, particularly with

reference to the time patterns of the headache attacks. Of special significance is the tendency of migraine headache to occur in some patients during periods of relaxation ("change of pace"), at nighttime, just before or with the menses, or in close-packed clusters lasting several weeks. Recognition of these features aids both in diagnosis and in therapy.

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27 Deering Street, Portland, Maine

#### Annual Meeting Dates For Your 1964 Calendar

Maine Medical Association, June 14-16, 1964 at The Samoset, Rockland, Maine.

American Medical Association, June 21-25, 1964 at the Fairmont and Mark Hopkins hotels and Civic Auditorium, San Francisco, Calif.



# Boston Medical Reports

MASON TROWBRIDGE, JR., M.D.

The Boston Medical Reports Committee is to be commended for its efforts to present to Maine physicians several important subjects by means of educational television. Assessing the extent to which this series will improve patient care is difficult. It is unfortunate that we cannot tag ideas with radioactive tracers and thus follow their course.

Never in history has the practicing physician been subjected to so much postgraduate medical education by high pressure gavage. The average practitioner resembles a nerve-muscle preparation; after a certain amount of stimulus he becomes refractory and no longer responds. If one desires a response one does not merely increase the voltage of the stimulus; one alters the environment of the preparation. Some factors that render a physician more or less responsive to new ideas will be discussed. These factors also affect the effectiveness of other methods of getting new ideas into the noggin of practicing physicians. To date everything short of trephining has been tried.

We have heard only four programs. The response has been mainly favorable, although there has been a considerable disparity of opinion on each. These programs were necessarily slightly pallid for the many listeners who had previously made rounds with or otherwise become acquainted with the speakers. Minimal personal contact with these teachers was far more memorable than the TV appearance. We missed Dr. Damashek's usual delicate sledgehammer whimsy. Criticism was often on picayune points, although if one L.M.D. asked me why Dr. Damashek didn't comment on pernicious anemia developing into the myeloproliferative syndrome, a dozen did. A telephone call to another hospital twenty-four hours after a program revealed that there had been no corridor or snack bar discussion of the program. The impact on Maine medicine of a few practitioners visiting university centers for a week or two of formal or informal training may be far greater than any series of TV programs.

The most popular presentation was that of Dr. Ingbar and Dr. Braverman. Many of us knew little about the usefulness of reserpine and the thyroid suppression test. It was also stated, "They did not talk down to us." The hypersensitivity of many practitioners to anything remotely resembling a patronizing attitude is one of the chief problems of postgraduate education. It can best be explained by the recently described "Town and Gown" syndrome. In no other field do professional men occasionally turn savagely on their former mentors. It is sometimes hard to believe that university physicians and private practitioners have a common adversary,

human suffering. There was isolated resentment of such statements as that it is advisable to order a serum bilirubin when confronted with jaundice. Yet I recently heard Dr. Stuart Finch tell an audience including many other university hematologists that in diagnosing iron deficiency the stained peripheral smear was useful. He did not produce an anaphylactic reaction among his listeners. Postgraduate medical teaching is difficult; the teacher encounters resentment if the subject is beyond the ken of his listener or if the listener already knows a particular fact. I was initially disappointed at not learning more of the newer knowledge of the staphylococcus and sensitivity testing. Yet on reflection it was apparent that the major problem of nosocomial staphylococcus infection will be solved not by such knowledge but by greater attention to the teachings of Semmelweis and Holmes.

Television and the traditional method of having a university teacher venture out to the hinterlands to address a group of doctors who have had too much to eat or are otherwise somnolent are of limited value until a more relaxed relationship between the university physician and practitioner exists. This will not be achieved until a more symbiotic relationship is achieved. The outlying physician must have the quiet confidence that he, too, is adding to our store of medical knowledge. University physicians should realize that if they wish to improve medicine nationwide, merely imparting newer ideas to existing practitioners is only a small part of the job. Encouraging dissemination of clinical and epidemiological investigation will make more American communities attractive to young physicians with an inquiring mind and avidity for new knowledge. Basic investigation should not be confined to a few "monasteries" as in the middle ages.

The Boston Medical Report programs are a bold and necessary experiment. Being personally responsible for a couple of local TV fiascos, I can appreciate their technical excellence. The charts were small, but I once had a chart on which the ink didn't show at all on TV. But currently postgraduate medical education can best be achieved by more personal contact with the university and by means of that dandy invention of Gutenberg.

This review may seem hypercritical, but my friends will attest that I am fundamentally a simple soul easily satisfied with the best of everything. Furthermore, it was strongly hinted that the Boston Medical Report group wanted someone to act as the devil's advocate. Let us hope that further opinion on the most enjoyable recent TV programs will appear in these columns.

# Maine Medical Association

## Program-in-Brief — 111th Annual Session

**The Samoset — Rockland, Maine**

**Sunday — Monday — Tuesday**

**June 14, 15, 16, 1964**

### **Sunday, June 14**

9:30 A.M. First Meeting of the House of Delegates

12:30 P.M. Luncheon

3:00 P.M. Second Meeting of the House of Delegates

6:30 P.M. Dinner

Speaker: DR. R. C. S. YOUNG, Birmingham, Michigan. Dr. Young appears through the courtesy of General Motors

Subject: **Meeting the Challenge of Leadership**

### **Hospital Administration Preparation for Disaster Casualties**

PHILIP K. REIMAN, Administrator, Maine Medical Center, Portland

### **Coordination of Medical Staff and Hospital Administration Plans and of Inter-Hospital Planning**

CHARLES W. STEELE, M.D., Internist and Cardiologist, Lewiston

11:35 A.M. Question and Answer Period

11:55 A.M. CARROLL P. HUNGATE, M.D. — Closing Summary

12:00 NOON to 2:00 P.M. Luncheon

1:30 to 4:00 P.M.

Scientific Program Sponsored by the Maine Chapter, American College of Surgeons

1:30 P.M. Business Meeting, Maine Chapter, American College of Surgeons

2:00 P.M. **Hypovolemic Shock** (Including discussion of low molecular dextran and review of some Swedish experiences)

GEORGE R. DUNLOP, M.D., Chief of Surgery, The Memorial Hospital, Worcester, Massachusetts

3:00 P.M. **The Emergency Treatment of Disaster Victims With Emphasis Placed on Therapy of Open Wounds and Other Major Traumatic Injuries**

CARROLL P. HUNGATE, M.D., Kansas City, Missouri

4:00 P.M. Election of President-Elect

6:00 to 7:00 P.M. Social Hour, Dutch Treat

### **Monday, June 15**

9:30 A.M. **Frozen Blood**

CHARLES E. HUGGINS, M.D., Instructor in Surgery, Massachusetts General Hospital, Boston, Massachusetts

10:15 A.M. to 12:00 NOON

**Emergency Medical Treatment of Disaster Victims.** Sponsored by the Maine Society of Internal Medicine and Medical Specialty Groups

10:15 A.M. **Medical Management of Disaster Victims during the Kansas City floods and after the tornado**

CARROLL P. HUNGATE, M.D., Kansas City, Missouri

11:00 A.M. **Panel: Medical Planning in Advance for Disaster**

Moderator, CARROLL P. HUNGATE, M.D.

**Hospital Staff Planning and Organization**

JOHN T. KONECKI, M.D., Radiologist, Lewiston



7:00 P.M. Annual Banquet

GOVERNOR JOHN H. REED

Speaker: DR. JOHN C. KRANTZ, JR., Professor and Head of Pharmacology, University of Maryland School of Medicine, Baltimore, Maryland

Subject: **The Simplicity to Wonder**

Presentation of Honorary Pins

## Tuesday, June 16

9:30 A.M. to 12:00 NOON

9:30 A.M. **Diagnosis and Management of Sigmoid Diverticulitis**

ALFRED HURWITZ, M.D., Portland

10:00 A.M. **Nutritional Regulation of Plasma Lipids and Lipid Metabolism**

LAURANCE W. KINSELL, M.D., Director, The In-

stitute For Metabolic Research, Highland-Alameda County Hospital, Oakland, California

11:00 A.M. **Pituitary Ablation in the Treatment of Diabetic Retinopathy**

CHARLES A. FAGER, M.D., Lahey Clinic, Boston, Massachusetts

12:00 NOON to 2:00 P.M. Luncheon

2:30 to 4:00 P.M.

Program Sponsored by the Maine Medico-Legal Society

2:30 P.M. **The Unwitnessed Homicide**

LESTER ADELSON, M.D., Pathologist and Chief Deputy Coroner, County of Cuyahoga, Cleveland, Ohio

6:30 P.M. Clam Bake

## Specialty Group Meetings

### Monday, June 15

2:00 to 4:00 P.M.

Section on Ophthalmology

Maine Society of Clinical Hypnosis

Maine Society of Obstetrics and Gynecology

Maine Society of Anesthesiology

### Tuesday, June 16

10:00 A.M. Maine Medico-Legal Society

Business Meeting

Presiding, IRVING I. GOODOF, M.D., Waterville

12:00 NOON Luncheon Meeting

Maine Radiological Society

2:00 to 4:00 P.M.

Maine Thoracic Society

Maine Society of Internal Medicine

Ear, Nose and Throat Group

Maine Chapter of the American Academy of Pediatrics

## House of Delegates

Included in the Order of Business for the meetings of the House of Delegates will be final action on the Budget for 1965, Standing and Special Committee Reports, Nominating Committee Report and matters presented at the Interim Meeting of the House of Delegates, published in this issue of the Journal, page 86.

## Election of Councilors

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 14 at 3:00 P.M.

Fifth District — Hancock and Washington

Sixth District — Aroostook, Penobscot and Piscataquis

## Sponsors

The speakers for the scientific programs are supported in part by grants from Eli Lilly and Company, Merck Sharp & Dohme and Maine Heart Association, Inc.

## Special Notices

### Election of President-Elect

The Election of a President-Elect will take place at the General Assembly, June 15 at 4:00 P.M.

## Golf Tournament

DANIEL R. SHIELDS, M.D., Chairman

# From the Secretary's Notebook

## Summary of Proceedings, Interim Meeting, M.M.A. House of Delegates, April 12, 1964 at Brunswick, Maine

The interim meeting was called to order at 2:15 P.M. by the President, Ernest W. Stein, M.D., (in the absence of the President-elect, Thomas A. Martin, M.D.), who turned the meeting over to the Speaker of the House, Linus J. Stitham, M.D.

There was a total attendance of 55, which included 31 delegates, 13 alternates, 7 councilors, the Secretary-Treasurer and three guests.

Financial Statement 1963 and Budget Proposed for 1965 – George E. Sullivan, M.D., Chairman of the Budget Committee presented a statement of Income and Expenditures for fiscal year ending December 31, 1963 and budget proposed for fiscal year ending December 31, 1965.\* Final action on the budget proposed for 1965 will take place at the annual meeting of the House of Delegates on June 14 at The Samoset, Rockland, Maine.

Proposed amendment to M.M.A. Constitution – On motion by George W. Wood, III, M.D. of Bangor, it was voted, almost unanimously, that the Fall Clinical Session be discontinued and Dr. Wood was instructed to send to the Association's office a recommendation that the M.M.A. Constitution, Article VII, Sec. 2, which follows, be amended to comply with this vote.

"Meetings of the Association may be called by the President, or by the Council, and shall be called by the President on petition of ten (10) members of the House of Delegates or fifty (50) members of the Association. There shall be more than one (1) scientific meeting sponsored by the Association each year."

A letter from Hadley Parrot, M.D., Secretary of the Penobscot County Medical Association states that the Executive Committee of the Penobscot County Medical Association met Tuesday, April 14. A quorum was present and the following motion was unanimously passed:

"Be it resolved that the Constitution of the Maine Medical Association, Article VII, Section 2 be changed so that the Association is not required to sponsor more than one scientific meeting each year."

1964 Annual Session program – Robert L. Ohler, M.D., Chairman of the Scientific Committee presented a resumé of the program planned for this session. The Program-in-Brief appears elsewhere in this issue of the Journal.

Negotiation of Medicare Contract – Daniel F. Hanley, M.D. stated that he had received a letter from the office for Dependent's Medical Care re this contract which will be coming up for renewal July 1, 1964. This letter requests that fees be reduced, in many instances this amounts to a 25% reduction (Rel. value decrease from average of \$4.13 to \$3.25 per point). (Copy of this correspondence has been sent to each county secretary and to members of the Health Insurance Committee.) On motion duly made and seconded, it was voted that the Committee and the Executive Director renegotiate the Medicare Contract to the best of their ability with the proper Government Agency.

\*Copy to Councilors, Delegates and Alternates with copy of Interim Meeting Summary on April 15, 1964.

Medical School for Maine – Charles A. Hannigan, M.D. stated that the Council of the M.M.A. has endorsed a Medical School Feasibility Study, that he had met with Dr. Lloyd H. Elliott at Orono and that an issue of The Journal of the M.M.A. devoted to a medical school in Maine has been discussed.

AMPAC-MEMPAC – Carl E. Richards, M.D. spoke briefly re this program to remind members that MEMPAC is still active, that there will be a request for money later and that he will have a more detailed report at the annual meeting in June.

M.M.E.F. – Dr. Hanley stated that the number of medical students from Maine has almost doubled since this fund was started. Over \$15,000 was loaned to medical students last fall and we expect to have requests for around \$22,000 next fall. The fund has approximately \$70,383 in cash and \$25,775 in loans; of this total of \$96,158, \$41,500 was received from members of the M.M.A. and the balance from outside sources. We are about one-third of the way to our goal and the program is going very well but we need the help of every member.

*Continued on Page 93*



**MAINE CANCER SOCIETY PRESENTS GRANT TO  
MAINE MEDICAL EDUCATION FOUNDATION —**

Armand Albert, M.D. of Van Buren, (left, front row) President of the Maine Cancer Society, presents a check for \$2,000 to Ernest W. Stein, M.D. of Pittsfield, President of the Maine Medical Association. Looking on as Dr. Stein receives this grant for the Maine Medical Education Foundation, are (left to right) Paul S. Hill, Jr., M.D., Saco, Councilor, M.M.A., Charles F. Branch, M.D., Lewiston, Councilor, M.M.A. and Daniel F. Hanley, M.D., Executive Director of the M.M.A.





DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### State-wide Vision Screening Program for Three-Year-Olds

ELLA LANGER, M.D.\*

With the approval of the Maine Medical Association, the Maine Chapter of the American Academy of Pediatrics, the Maine Chapter of the American Academy of General Practice and the Maine Optometric Association, three state health divisions and other units of the Department of Health and Welfare are starting a State-wide visual screening program for all 3-year-old children in Maine during the month of May. The purpose is to prevent amblyopia, which, literally translated, means "dull eye," a condition which is estimated to affect 4% of the general population. This means that of the approximately 20,000 children born annually in Maine, 800 can be expected to develop irreversible vision loss in one eye unless this condition is sought out, detected and treated early.

Departmental divisions cooperating in the program are: The Division of Maternal and Child Health, Division of Public Health Nursing in the Bureau of Health and The Division of Eye Care and Special Services in the Bureau of Social Welfare. They will have the assistance of the Office of Health Education and the Division of Research and Vital Records. Key officials comprising the departmental committee directing the program are: the Commissioner ex-officio; the author of this article (Dr. Langer, Director of Maternal and Child Health); Mary M. Sullivan, R.N., Director of Public Health Nursing; Ruth T. Clough, M.Sc.H., Health Education Consultant; Edson K. Labrack, M.P.H., Director, Division of Research and Vital Records and C. Owen Pollard, M.S.W., Director, Division of Eye Care and Special Services.

A State Advisory Committee has been named as follows: Richard H. Dennis, M.D., Ophthalmologist, Waterville; Malcolm W. Cass, O.D., Optometrist, South Portland; Henry Thacher, M.D., Pediatrician, Lewiston and John Denison, M.D., General Practitioner, Gardiner.

The preliminary screening test will be carried out by providing parents, guardians or anyone in loco

parentis with a simple subjective visual screening test kit which may be used at home under ordinary conditions. Kits, complete with detailed instructions, a cardboard occluder (or test frame) and a postage paid card on which to report the results of the preliminary screening will be mailed to the home addresses of all 3-year-old children whose births are registered at the Department's Division of Research and Vital Records. Parents and guardians of 3-year-olds who were not born in Maine may obtain the home screening kits by writing their request to the Division of Maternal and Child Health, Department of Health and Welfare, State House, Augusta, Maine.

Cards which indicate that a child either sees from one eye better than the other or that the parent or guardian is unable to determine if there is a difference or not, after being returned to the Division of Maternal and Child Health, will be processed through the statistical machinery of the Division of Research and Vital Records. Plans will then be made for a re-test in the area of the individual's home.

Planning details for the re-testing to be carried out by volunteers trained for the purpose by qualified professional persons aided by staff nurses of the Division of Public Health Nursing will be announced later and parents of children who require re-testing will be notified in ample time. Cases discovered through this final screening method to be in need of evaluation by a specialist will be referred by the Department unless the family can provide this service.

It is expected that salient points of the program will be refined and tested continually as it proceeds and that the program itself can become an integral part of general child health supervision and child health procedures for Maine children.

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An announcement of Preliminary Plans For New England Health Institute appears on Page 94.

\*Director, Division of Maternal and Child Health

## *Maine Heart Association Notes*—————



### **Present Status of Thrombolytic Therapy**

“Despite the extraordinary clinical potential of thrombolytic therapy, the many investigations performed with thrombolytic agents in man, and the release of several thrombolytic drugs for general use, many problems remain to be solved before these agents can be utilized on a practical clinical basis. Only a few controlled studies have proved that blood clots can be dissolved in man, and these required such complex laboratory control that the use of thrombolytic agents under any but the most elaborate research conditions is precluded. . . .

“There are many problems to be solved in therapy with thrombolytic agents. It is important to recognize that even after a particular agent has, in fact, been shown to be thrombolytic under the best controlled ‘laboratory conditions,’ the same agent must be reevaluated for its clinical efficacy, using simple laboratory tests for the control of routine therapy under practical conditions. Only at this time will it be possible to begin to determine the true clinical usefulness of thrombolytic therapy, with appropriate, well-designed clinical trials.”

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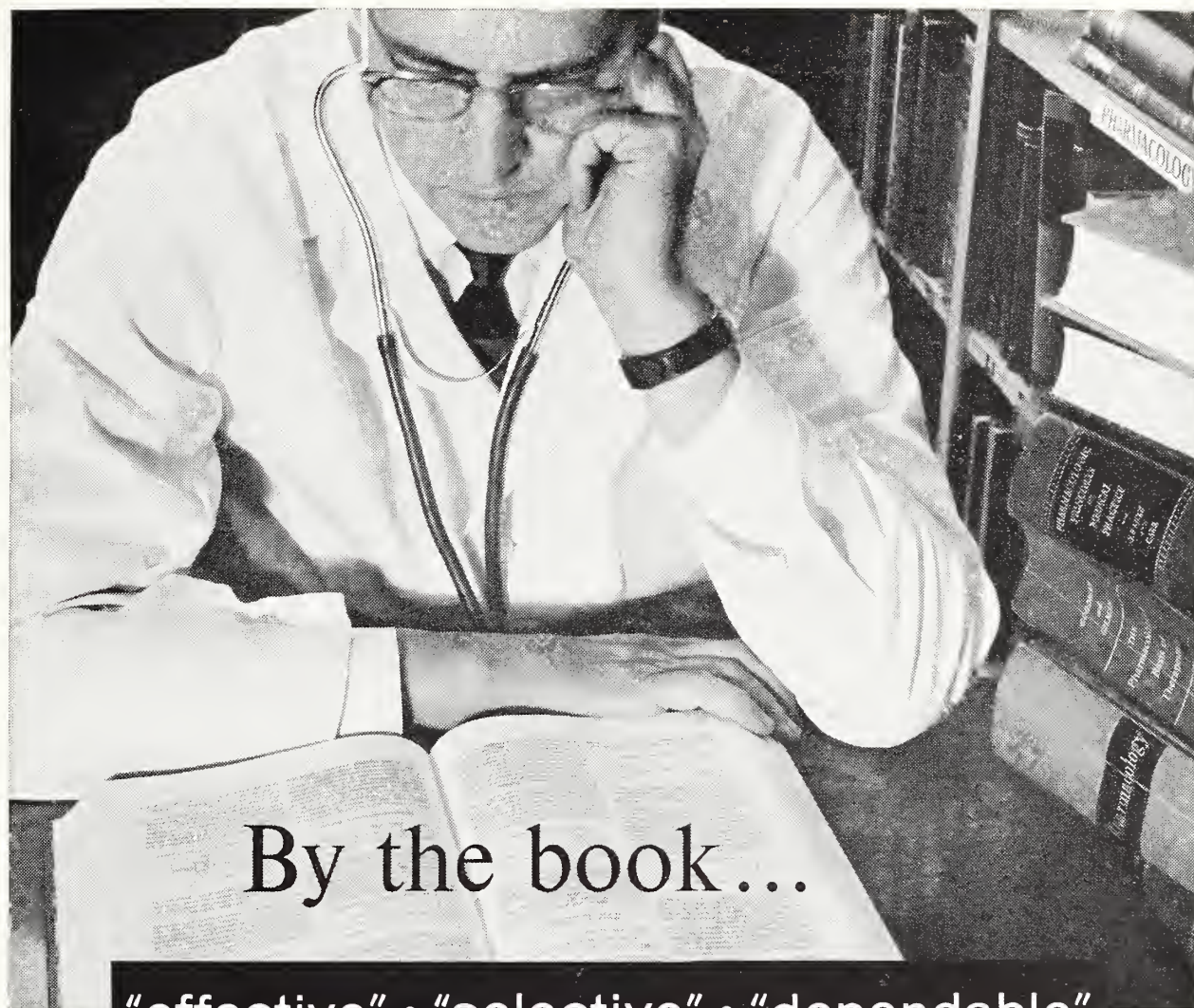
Reference: JOHNSON, Alan J.: American Heart Journal, Vol. 67:418-420 (1964)

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### **“What Everyone Should Know About Smoking and Heart Disease”**

Above is the title of a leaflet issued this month by the American Heart Association to answer many of the layman’s questions concerning the dangers of smoking with special emphasis on the heart and circulatory diseases. The leaflet is available to physicians for distribution to patients and for waiting room display. The publication is a service of the Maine Heart Association supported by the Heart Fund. The leaflet can be obtained, free of charge, by writing the Maine Heart Association, 116 State Street, Augusta, Maine.





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## Necrology

ERNEST D. HUMPHREYS, M.D.

1883-1964

Ernest D. Humphreys, M.D., 80, of Pittsfield, Maine died on March 15, 1964. He was born in Brownville, Maine on December 22, 1883, son of John and Mary Louise Humphreys. He graduated from Brownville High School and did postgraduate work at Higgins Classical Institute. He attended Bowdoin College and graduated from Bowdoin Medical School in 1910.

Dr. Humphreys began the practice of medicine as assistant surgeon for the Canadian Pacific Railroad, did general practice in Jackman for 28 years and then moved to Pittsfield where he practiced for 24 years until his death.

Dr. Humphreys was an Honorary member of the Maine Medical Association and the Somerset County Medical Society, having received a 50-year pin in 1960. He was also a member of the American Medical Association and the New England Hospital Service.

He was a special medical examiner for the armed forces during both World Wars. For many years, he was county medical examiner in Jackman, school physician for the local schools and the Maine Central Institute. He was trustee of the Jackman Congregational Church, treasurer of the Library Association and served on the school board for 20 years. He was a charter member of the Pittsfield Kiwanis Club, a member of the First Congregational Church, a 32nd degree Mason, a member of the Kora Temple of Lewiston, president of the Sebasticook Valley Hospital staff at Pittsfield and was past chief of staff of the Scott-Webb Hospital at Hartland.

Surviving are his widow, Elizabeth; one daughter, Mrs. John W. Crosson of Exeter, New Hampshire; one grandson, Martin E. Bunker of Pittsfield; one sister, Mrs. Charles Eastman of Auburn and several cousins.

## County Society Notes

### PENOBSCOT

A meeting of the Penobscot County Medical Society was held at the Bangor House in Bangor, Maine on February 18, 1964, with the President, William A. Purinton, M.D., presiding. Thirty-five members and guests were present.

Lester F. Soyka, M.D., a Research Fellow at the Massachusetts General Hospital, was the guest speaker. Dr. Soyka discussed Therapy of Short Stature in Children With Particular Emphasis on the Use of Pituitary Extracts.

The following members of the Penobscot County Medical Society have been appointed as Delegates to the 1964 Maine Medical Association House of Delegates: Drs. Lloyd Brown, Leonard G. Miragliuolo, Carl E. Blaisdell and Arthur N. Lieberman, all of Bangor; and George W. Wood, III of Brewer. Alternate Delegates: Drs. Charles D. McEvoy, Jr., Clement S. Dwyer, George O. Chase and William M. Shubert, all of Bangor; and Irvin E. Hamlin of East Millinocket.

HADLEY PARROT, M.D.  
*Secretary*

### KENNEBEC

A meeting of the Kennebec County Medical Association was held on February 20, 1964 at the Pioneer House in Augusta, Maine.

The President, Kenneth W. Sewall, M.D., presided at the business meeting.

Ernest W. Stein, M.D., President of the Maine Medical Association, greeted the County Association and told of his recent attendance at the New York State Medical Society Convention.

Alta Ashley, M.D. introduced Mr. William M. Kitching, a member of the venereal disease section of the Division of Communicable Disease Control of the United States Public Health Service. Mr. Kitching spoke briefly of the intensified efforts on the part of the United States Public Health Service in eradicating syphilis. He also spoke of the necessity of reaching all sexual contacts and in thorough follow-up care.

The clinical portion of the program was a panel discussion of the emergency management of crash injuries. Lucien F.

Veilleux, M.D. of Waterville moderated the panel, which consisted of Jose M. Rodriguez, M.D., neurosurgeon; H. Richard Hornberger, M.D., thoracic surgeon and Loring W. Pratt, M.D., otolaryngologist. Dr. Rodriguez showed the number of neurological injuries in a statistical way and spoke of their evaluation by clinical means. Dr. Hornberger appealed for the attending physician to have the will to do something active in the treatment of chest injuries where action is necessary. Dr. Pratt illustrated the technique of emergency tracheostomy and illustrated his points with examples of the tracheostomy saving lives or the failure of tracheostomy in losing lives. Dr. Veilleux appealed for a decent emergency cabinet in every emergency room which was clearly marked and well stocked. He also appealed for a single commander in the care of each critical injury saying that several people of responsibility never worked well. He then enumerated the diagnostic steps and the proper sequence of events in caring for the injured patient.

EARLE M. DAVIS, M.D.  
*Secretary*

### CUMBERLAND

A meeting of the Cumberland County Medical Society was held at the U.S.S. Maine Room of the Portlander in Portland, Maine on February 20, 1964, with eighty-five members and guests present.

The President, Charles R. Geer, M.D., presided at the business meeting. A letter was read from Mr. Robert Moore of the Governor's Executive Council emphasizing the importance of our writing letters about impending political questions.

Boris A. Vanadzin, M.D. reported on the Health Career questionnaires, stating that he had received 70 replies of which there were 28 persons indicating willingness to take one or more selected high school students during one day of April vacation. John F. Gibbons, M.D. read the roster of physicians responsible for medical recruitment in various schools and communities.

Dr. Geer reported that the liaison committee with the



bar association had been appointed, consisting of Dr. Robinson L. Bidwell, Chairman, Dr. George F. Sager and Dr. John A. Godsoe. It is expected that this group can meet with a similar group from the bar association to attempt to solve problems which have heretofore been distressing to both groups.

Mr. Michael J. Maroon of the Internal Revenue Service was introduced by Dr. Geer. Mr. Maroon discussed the source of and expenditure of the income tax, the physical make-up of the Internal Revenue Service, the manner of processing an income tax return, the ethics and integrity of the agents and points drawing auditors' attention to specific tax returns.

A meeting of the Cumberland County Medical Society was held on March 19, 1964 at Valle's Steak House in Portland, Maine, with 105 members and guests present.

The meeting was called to order by the President, Charles R. Geer, M.D., following a social hour and dinner.

A resolution was read by Daniel F. Hanley, M.D., Executive Director of the Maine Medical Association, on the death of Charles H. Patton, Jr., M.D. A committee was appointed to prepare a resolution on the death of Luther A. Brown, M.D.

Announcement was made of the Civil Defense Emergency Hospital which is touring the state.

U. S. Representative Clifford G. McIntire, was introduced by Dr. Geer. Rep. McIntire discussed in a general way his background and his feelings about the economics of medicine, indicating his opposition to the administration supported program.

STANLEY B. SYLVESTER, M.D.  
*Secretary*

#### WASHINGTON

A regular meeting of the Washington County Medical Society held on March 4, 1964 with a dinner at the Sunset Restaurant in St. Stephen, New Brunswick, was attended by twenty-nine members and guests.

This was followed by a meeting at the Calais Regional Hospital with nineteen members and guests attending.

The meeting of the Washington County Society was presided over by James C. Bates, M.D. of Eastport, Maine. Ernest W. Stein, M.D., President of the Maine Medical Association, spoke regarding recent work of the State Association.

Paul H. Pfeiffer, M.D. of Waterville spoke on the Maine Medical Education Foundation and the need for further contributions to build up the fund. So far the fund has been used to good advantage and the number of students going into Medical College has shown a moderate increase.

The scientific portion of the program was presented by a panel from the Central Maine Sanatorium of Fairfield, Maine headed by William B. Grow, M.D. The panel included Drs. H. Richard Hornberger, John F. Reynolds, Paul H. Pfeiffer of Waterville and Percy McIntire a member of Dr. Grow's staff at the Sanatorium. The panel took up various aspects of medical and surgical control of tuberculosis, illustrating their remarks with x-rays.

Following the meeting, members and their wives were entertained at the home of Dr. and Mrs. Hazen C. Mitchell of Calais.

KARL V. LARSON, M.D.  
*Secretary*

#### YORK

The York County Medical Society met on March 11, 1964 at the Webber Hospital in Biddeford, Maine. Twenty-five members and two guests were present. Following a social hour

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and dinner, the meeting was called to order by the President, Roger J. P. Robert, M.D.

Willem Nieuwkerk, M.D., Psychiatrist located at Kennebunkport, Maine, was the guest speaker. He gave a very interesting talk on Family and Child Guidance Assistance, followed by a very interesting question and answer period.

The remainder of the evening was taken up with a discussion of nursing home care.

CHARLES W. KINGHORN, M.D.  
*Secretary*

#### ANDROSCOGGIN

The monthly meeting of the Androscoggin County Medical Society was held at the St. Mary's General Hospital in Lewiston, Maine on March 26, 1964. There were twenty members present and the President, Robert D. Wakefield, M.D., presided. The sub-committee approved at the February 1964 meeting, to investigate the need for and to obtain Visiting Nurse Services as appointed by Dr. Wakefield consists of Drs. Charles A. Hannigan, Rudolph Haas and Henry C. Thacher.

Communications consisted of a letter from Peter Bowman, M.D., Superintendent of the Pineland Hospital, requesting information as to the needs in this county for chronically ill patients not requiring institutionalization in a general hospital; how these needs are being met at present; and how local physicians can be used to help plan appropriate facilities in caring for these patients. During the discussion that followed, there was concern expressed for people in some nursing homes, who for years receive only token medical care at best.

A guest at this meeting was Ernest W. Stein, M.D., President of the Maine Medical Association. Dr. Stein mentioned

the existence and activities of the Committee chaired by Dr. Hannigan to conduct a survey as to the need for a medical school in Maine; briefly presented the proposed 1965 budget for the Maine Medical Association; expressed concern over the loss of drug advertising in the JMMA; stated that in addition to existing TV and radio medical education activities, the Albany Medical Group is planning a medical TV education program in the future, and mentioned the possibility of a Maine State Malpractice Group Insurance Policy which is at present being investigated by Alexander M. De la Garza, M.D. Dr. Stein suggested that the members of the county society thank those pharmaceutical house representatives whose products are advertised in the Journal and urge those who do not advertise to do so.

The Memorial Testimonial to Richard N. Goldman, M.D. as prepared by John A. James, M.D. was read, accepted by the society, and it was voted that copies be sent to Dr. Goldman's parents, wife, and to the Secretary of the Maine Medical Association.

Dr. Wakefield directed the nominating committee to present nominations at the April county society meeting to fill Dr. Goldman's unexpired term as Councilor.

The guest speaker was Mr. Henderson Dudman who spoke on The Doctor and His Public Image.

RONALD S. POTTS, M.D.  
*Secretary Pro tem*

#### New Member

#### CUMBERLAND

Bernard Givertz, M.D., 131 Chadwick St., Portland

## Book Reviews

### **Patient Care and Special Procedures in X-Ray Technology — By Carol H. Vennes, R.N., B.S. and John C. Watson, R.T., Second Edition. Published by C. V. Mosby Company, St. Louis, Mo.**

The second edition of this text book contains 220 pages and is liberally illustrated.

This volume fulfills a definite need for student x-ray technicians, and should be well received as was the first edition. It contains chapters which describe relationships of x-ray technicians to patients, to radiologists, to nurses and to other hospital personnel. Receiving particular attention are those "nursing procedures" which are used daily by the x-ray technician in her work or as a member of a medical team. The book is well divided into chapters, each dealing with a facet of special patient care as it relates to the x-ray technician.

JOHN F. GIBBONS, M.D.  
Portland, Maine

### **Physical Diagnosis. The History and Examination of the Patient — By Drs. John A. Prior and Jack S. Silberstein, Second Edition. Published by C. V. Mosby Company, 455 Pp with 277 illustrations.**

Drs. Prior and Silberstein have assembled a beautifully written text on physical diagnosis. They author the sections on medical history, general inspection, head, face, neck, thorax, lungs, cardiovascular system and extremities; while others have written sections on the mental examination, ears, nose and

throat, eyes, breast, abdomen and male genitalia, female genitalia, motor skeletal system and pediatric examination.

The authors' deft hands are especially enjoyed in the medical history, which places special emphasis on the thorough, astute review of systems, past history and objective evaluation of the present illness. Much emphasis is placed on avoiding vague historical statements, self diagnoses and muddled thinking.

The sections on the examination of the eyes, ears, nose and throat are superb. They are lucid, detailed, and practical. Many simple drawings and photographs illustrate difficult details. There are little gems like how to detect a shallow anterior chamber, properly determine intraocular tension by finger tension and how to use the corneal light reflex to detect deviation of an eye.

The section on the chest is most lucid. Gone are the crepitant, and subcrepitant rales, squeaks, gurgles and rattles and their cacophony of confusion. In their place are rhonchi and rales, the latter divided into fine, median, and coarse; the former into sibilant and sonorous. Normal breath sounds are described as vesicular, bronchovesicular and tracheal.

The examination of the breast is conducted in a thorough fashion with many illustrations. On the other hand the cardiovascular system, despite its clarity, lacks in part modern terminology. Murmurs are not described as pan-systolic or diamond shaped but the author does not miss ejection clicks, protodiastolic and presystolic gallops.

The abdomen is divided into 4 quadrants. When I went to school, we had to learn 9. I missed the epigastrium but altogether found 4 quadrants more logical and simpler.

While many textbooks on physical diagnostic gallop through



or even spurn the female genital tract, this textbook devotes an entire thorough section, beautifully illustrated to this.

The section of the extremities has good appropriate photographs and is well written. It suffers a little from excess brevity. It should be read in the same sitting with the subsequent chapters on neurology and motor skeletal system. At some future date these could be combined into one large section as there is some overlapping. The description on the back needs expansion.

A pediatric examination, carefully and clearly written, completes this delightful book.

The textbook is heartily recommended for students and all clinicians for learning and review. It is fun to read, at times humorous, at all times lucid and thorough.

PETER F. LANSING, M.D.  
Augusta, Maine

5th District — Russell G. Williamson, M.D., Blue Hill

6th District — John B. Madigan, M.D., Houlton

The report of the Nominating Committee shall be the first Order of Business at the Second Meeting of the House of Delegates on Sunday, June 14 at 3:00 P.M.

Election of Councilors — Announcement was also made that the terms of the Councilors from the Fifth and Sixth Districts will expire in June and that in accordance with the By-Laws, "Nominations for members of the Council for any District where there is a vacancy shall be made by a *caucus of the members of the House of Delegates of that District*. Each candidate for Councilor must be a resident of the District for which he is nominated."

Election of Councilors will take place at the Second Meeting of the House of Delegates on Sunday, June 14 at 3:00 P.M.

Adjourned at 4:25 P.M.

INTERIM MEETING, M.M.A. HOUSE OF DELEGATES

*Continued from Page 86*

Legislative definition of medical records in a hospital — Roger J. P. Robert, M.D., who requested a legislative definition of medical records in a hospital, will send in a resolution re this subject for presentation at the June meeting.

Nominating Committee — Announcement was made of the following members appointed by Thomas A. Martin, M.D., President-elect, to serve on the Nominating Committee:

- 1st District — John F. Gibbons, M.D., Portland — Chairman
- 2nd District — Morris E. Goldman, M.D., Lewiston
- 3rd District — Harry G. Tounge, Jr., M.D., Camden
- 4th District — Samson Fisher, M.D., Waterville

CORRECTION

Clinical Diagnosis and Management of Hepatitis Patients — In this paper by Drs. Heinz F. Eichenwald and Henry R. Shinefield published in the April, 1964 issue (Vol. 55, page 67), paragraph 1, line 2, left hand column, should have read "of permanent residua following hepatitis is."

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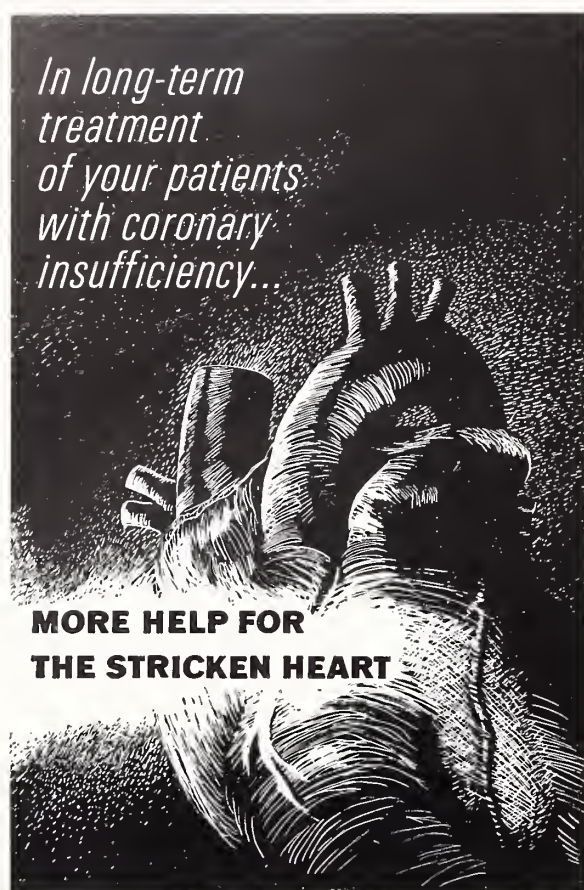
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## **Preliminary Plans for New England Health Institute**

The 30th annual New England Health Institute will be held June 17 through June 19 at the Griswold Hotel, Groton, Conn., Franklin M. Foote, M.D., Commissioner Connecticut State Department of Health, has announced. Participants will be more than 500 professional workers in the field of public health in all six New England states. Among the subjects to be presented by prominent public health authorities will be: Advances in Communicable Disease Control, Environmental Health, Newer Laboratory Procedures and Developments in Out-of-Hospital Care.

The New England Health Institute was originated in Connecticut in 1922. In that year, John T. Black, M.D., then Commissioner of Health, and Stanley H. Osborn, Deputy Commissioner, felt there was need for a regional meeting for the New England states on the latest scientific information and viewpoints relating to public health. As a result, on May 1, 1922, the first New England Health Institute was held at Hartford. Since 1922, with the exception of the depression and war years, NEHI has rotated through most of the New England states on an annual basis. The last Institute held in Connecticut was in 1958.

Milton Geyer, Chief, Public Health Education Section, Connecticut State Department of Health, and Chairman of the Institute, has appointed five members of the department as Committee chairmen. They are: Mila E. Rindge, M.D., Director, Regional Office, Northeastern Connecticut, Local Arrangements; Laurence A. Fagan, Chief Administrative Services Officer, Finance; Judith A. Flynn, Public Health Education Consultant, Publicity; John S. Pullman, Public Health Education Consultant, Exhibits; and Edwin T. Tracy, Chief, Public Health Statistics Section, Registration.

Miss Ruth Clough, M.Sc.H., Health Education Consultant for the Maine Department of Health and Welfare is serving as liaison person and will have additional information later on the final plans for the forthcoming Institute.

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# The Journal of the Maine Medical Association

Volume Fifty-Five

Brunswick, Maine, June, 1964

No. 6

## Bilateral Papilloma of the Choroid Plexus\*

H. S. BAAR, M.D., PH.D. and J. GALINDO, M.D.

Papillary tumor of the choroid plexus was first described by Guerard in 1833<sup>7</sup> in the right lateral ventricle of a three-year-old girl. Although this tumor is well-known it is a relatively uncommon type of intracranial neoplasm.

In the series reported by Cushing<sup>3</sup> in 1932, papillomas of the choroid plexus comprised only 0.5% of all intracranial tumors. Zulch,<sup>31</sup> 24 years later, arrived at an identical figure of 0.5 to 0.6%. Similarly, Sandison (1956)<sup>23B</sup> found two examples in 9700 necropsies which would correspond, roughly, to one case in 500,000 hospital admissions.

Ingraham and Matson,<sup>11</sup> on the other hand, reported in 1954 that, in pediatric clinics, papilloma of the choroid plexus accounts for up to 3% of all brain tumors encountered.

### CASE REPORT

K. T., born at full term in October of 1957 from healthy parents. She was the oldest of three children, the other two siblings normal and healthy. The mother suffered no illness during pregnancy. Delivery was at full term; labor proceeded normally; elective application of low forceps was employed.

The birth weight was 8 pounds, 13 ounces; the head circumference was 17 inches. The child appeared to do well until the onset of spells at the age of three months. The latter consisted of staring, jerking of the arms, more on the left than on the right, and sometimes of the legs. These episodes lasted a few seconds and occurred but once or twice on some days, while on other days they were noted as many as 15 or 20 times a day. On one occasion during a spell, nystagmus was noted. The patient has never walked nor talked.

In March of 1958 the patient was admitted to the Maine Medical Center for a period of six days, at which time an electroencephalogram was taken which was reported as showing hypsarrhythmia. A subdural tap was done which was reported as being negative. The patient was placed on phenobarbital and was referred to the Childrens' Medical Center in Boston. While

at that institution, a spinal tap was done which showed a clear cerebrospinal fluid with an opening pressure of 100 mm. Pandy was 2+, sugar 48 mg%, chlorides 123 mg%, protein 120.8 mg%. The cell count showed 2 monocytes per cubic mm. Cultures were negative. Two urine samples were examined for cytomegalic inclusions and were reported as negative. However, in the second sample there were a few polymorphonuclear leukocytes. Two catheterized urine specimens were cultured for virus, both samples were negative. At this time the head did not transilluminate; the anterior fontanel was open. There was an equivocal transient nystagmus on lateral gaze. In both optic fundi there were large areas of white scarring, bordered by black pigment. The ophthalmologist thought that these were consistent with developmental defects, but could conceivably represent an infection with toxoplasma. On her fifth hospital day she developed right hemiparesis which involved the entire right half of the body. The deep tendon reflexes were more active on the right, and an electroencephalogram showed diffuse cerebral dysrhythmia and cortical dysfunction with a slight suggestion that the right side was more involved than the left. The patient was discharged with the following diagnoses: 1. Myoclonic state 2. Mental retardation 3. Toxoplasmosis? congenital? 4. Chorioretinitis from toxoplasmosis? . . . Developmental? . . .

At the age of four years, the patient was again seen at the Childrens' Hospital in Boston. At this time, the head circumference was 55 cm. Bilateral Babinski was present. The toxoplasma dye test on the mother was positive in low dilution  $\frac{1}{4}$  and was negative in the baby. The skin test on the mother was also positive.

She was admitted to the Pineland Hospital and Training Center on February 21, 1963. At this time she was obviously hydrocephalic with a head circumference of 65 cm. She was unable to roll over, stand, walk, or talk. While in the supine position, the postural attitude consisted of external rotation of the limbs with flexion of the elbows and knees. There was a claw-like attitude of the fingers bilaterally, and there was generalized hypotonus.

There was complete lack of coordinated movements and divergent strabismus was noted bilaterally to the left.

The plantar reflexes were bilaterally flexor and the sensory system was apparently normal.

She cried if disturbed and, although she was never on anti-convulsant therapy in the hospital, she never had any convulsions. She drank only strained foods from a bottle which she made no attempt to hold with her hands.

\*Presented by Drs. Baar and Galindo, of the Department of Pathology at Pineland Hospital and Training Center, at the annual meeting of the Pineland Medical Staff on June 5, 1963.





FIG. 1. This view shows the under surface of the brain. The lateral ventricles have been opened and the papillary choroidal tumors are visible in each lateral ventricle.

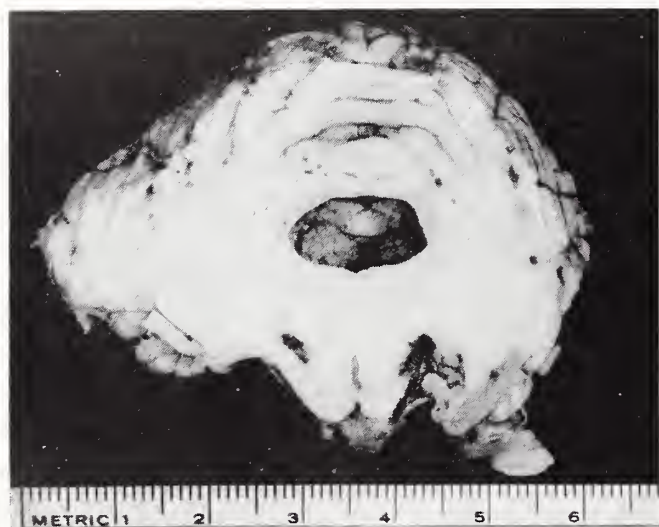


FIG. 2. The fourth ventricle shows marked dilatation and through it, the hyperplastic choroid plexus is visible.

Her serology was always negative for syphilis and the Berry and Dorfman-Lorincz tests for chondroitinsulphuric acid were negative. X-rays of the skull showed marked hydrocephalus with the anterior and middle fossae dilated and the parietal bones thinned. The occipital fossa was small and there was some suggestion of cerebellar depression. The radiologist thought that these findings were consistent with hydrocephalus due to obstruction of the aqueduct of Sylvius.

Three days before death she developed fever which, on one occasion, rose to 102° F. She had a white count of 17,250; hemoglobin of 16 grams percent; red cell count of 5,320,000 which was attributed to her dehydration; and a differential count of 70% segmented neutrophils and 8 band forms. A smear from the tongue showed *Candida Albicans* and a few polymorphonuclear neutrophils. She expired April 13, 1963.

#### POST MORTEM EXAMINATION

At autopsy, the body was that of an emaciated child with an enormous head that measured 65 cms. in circumference. The immediate cause of death was bronchopneumonia. The bones of the calvarium were extremely thin, 1 to 2 mm. in thickness, and contained unsharply demarcated areas of translucency. The dura mater and its venous sinuses were normal. The brain was

transformed into an extremely large, thin-walled sac. The thickness of the brain substance over the lateral ventricles was only 1 mm. The leptomeninges were normal. The lateral ventricles were extremely dilated. In the left one, at the site of the choroid plexus there was a soft mass, 6.5 x 4 x 2 cm., pinkish-gray in color with pinhead-sized white areas, and a surface which resembled that of a cauliflower. A smaller tumor of exactly the same appearance, 3.5 x 3 1/4 x 1 cm. was present in the choroid plexus of the right lateral ventricle. (Fig. 1)

Both foramina of Monroe were patent and were 1 cm. in diameter. The third ventricle, Sylvian aqueduct, and the fourth ventricle were markedly dilated, but considerably less than the lateral ventricles.

The foramen Magendie was widely patent. The foramina of Luschkae were patent and normal in size; the weight of the brain, drained of the cerebro spinal fluid, was 1040 grams. The fourth ventricle was wide, and its choroid plexus hyperplastic. (Fig. 2)

The first impression was that of a purely hyper-secretory hydrocephalus. However, meticulous dissection revealed a thin, translucent membrane, tightly stretched transversely across the Sylvian aqueduct, completely obstructing the lumen. (Fig. 3)

Histologically, the tumor mimicked the normal choroid plexus and was composed of lax delicate connective tissue stalks, rich in vascularity, which were peripherally capped by a layer of non-ciliated basophilic epithelial cells of the cuboidal or columnar types which, in places, were in a single cell layer, while in others they were haphazardly disarranged and occurred in 2 or more layers. (Fig. 4)

A number of sections of the tumor were stained with Phosphotungstic acid hematoxylin and did not reveal any presence of blepharoplasts or of cilia.

Several of the tumor sections showed well-formed Haversian bone. (Fig. 5)

No herniation of the cerebellum through the foramen magnum was present.

We found no evidence of cytomegalic inclusions in the salivary glands or in any of the other organs.

Calcifications were strictly confined to the choroid plexus and were not evident in the brain, either on gross or microscopic examination, or in a post mortem X-ray examination of the brain.

#### DISCUSSION

Papillary tumors of the choroid plexus are generally benign and occasionally malignant tumors of cauliflower-like appearance which vary in color from whitish-gray to gray-pink, and occasionally are reddish in color. These tumors grow, as their name implies, from the choroid plexus, and although well limited most of the time, they are not encapsulated. They are richly vascularized and at surgery or spontaneously are capable of considerable bleeding.

These papillary tumors, like the choroid plexus itself, are located within the cerebral ventricles and, although they may be located in any ventricle, 50 to 60% of them occur in the fourth ventricle. The location of the tumor in both lateral ventricles, as present in our case, is extremely rare. (Laurence)<sup>13</sup>

Papillary tumors of the choroid plexus, as mentioned before, simulate the normal choroid plexus and, therefore, histologically show a branching stalk of delicate connective tissue fibers with an abundant vascular supply and a peripheral crown of cuboidal to columnar epithelial cells in a single layer. On occasions like in the present case, the cells may show some disarrangement



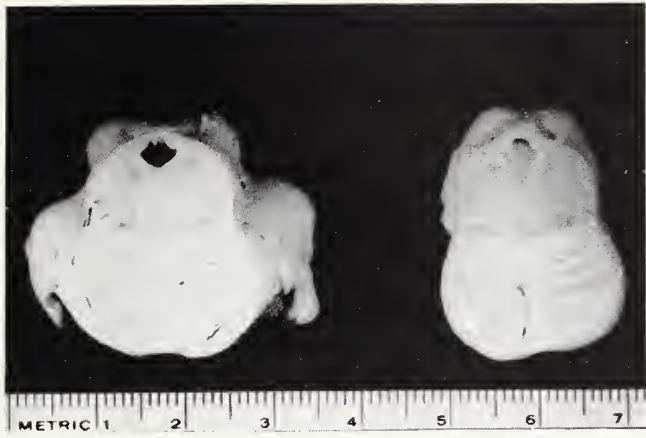


FIG. 3. Cross-sections of the pons, the one on the left showing a patent lumen, the one on the right demonstrating obstruction by a tense, delicate, translucent membrane.

and may appear in more than one cell layer thickness; this should not be construed as evidence of malignancy, for the latter diagnosis can only be made when true invasion of surrounding structures occurs.

The epithelial cells each have a large spherical nucleus, usually located near the base of the cell, and the cytoplasm is finely granular.

Differentiation between a papillary choroidal tumor and an ependymal tumor must often be sought on a histologic basis; features to look for are cilia and blepharoplasts which occur in ependymal tumors but which are not present in papillary tumors of the choroid.

Another distinguishing feature is that ependymal cells rest on glial tissue, whereas papillary tumors show the already described epithelial cell layer resting on connective tissue stalks.

Apart from their rarity, the interest in these tumors stems from the role in the pathogenesis of hydrocephalus. Herren<sup>10</sup>, in his study of over 80 cases, concluded that hydrocephalus is an almost universal accompaniment of the papillomas of the choroid plexus. This is, of course, at variance to the statement made by Dorothy Russell in her monograph on hydrocephalus 1954<sup>22</sup> in which she says, "It may be asked why an associated hydrocephalus accompanies only about 50% of the papillomas of the lateral ventricle."

The associated hydrocephalus in those afflicted with this variant of intracranial neoplasm serves as an interesting diagnostic guidance point, for on many occasions, and particularly in children, hydrocephalus is the only clinical feature present.

Suspicion of the diagnosis of this condition must be entertained by the physician for, in spite of the rarity, this process constitutes one of the few types of intracranial tumor which, when surgically removed, is capable of resulting in a successful cure.

The presenting symptoms might be those of hydrocephalus; headache, ocular difficulties, etc., regardless of the underlying etiology of the hydrocephalus itself, therefore, the importance of ventriculography in establishing the diagnosis.

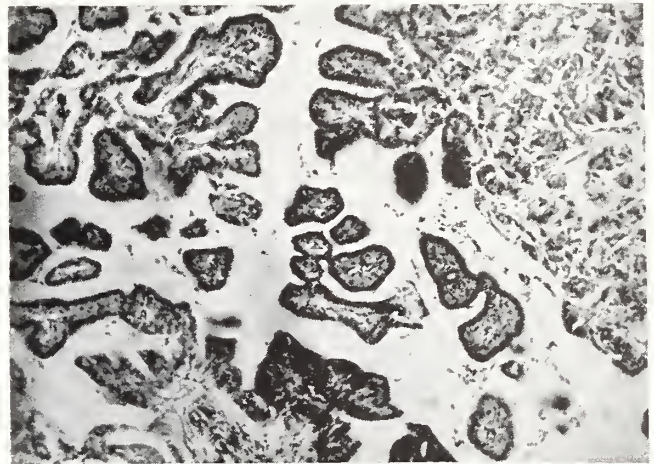


FIG. 4. A section of the tumor showing delicate connective tissue stalks with capillaries visible. There is a peripheral cap of epithelial cells covering the stalks. On the right upper quadrant of the photograph some disarrangement of cells is noted. H. E. x/1/100.

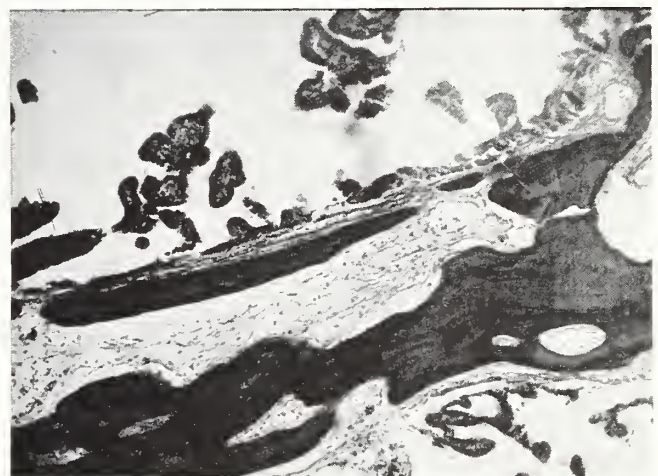


FIG. 5. Within the papillary stalk of the tumor, spicules of well-formed Haversian bone are evident. H. E. x 400.

The production of cerebrospinal fluid has been assumed by some to be due to diffusion of cerebrospinal fluid through the ependyma. However, the present consensus of opinion is that cerebrospinal fluid is formed by a process of active transport by the choroid plexus.

If the above is really the case, it should not be difficult to visualize over-production of cerebrospinal fluid in a papilloma of the choroid plexus which has resulted in an increase of many times the excretory surface of the choroid plexus.

The circulatory pathway of cerebrospinal fluid is through the foramina of Magendie and foramina of Luschka into the cisterna cerebello-medullaris and from there into the subarachnoid space over the cerebral hemispheres, to be absorbed by the arachnoid villi including the granulations of Pacchioni, then emptied into the superior longitudinal sinus.

Evidence that papillary tumors of the choroid plexus are truly capable of overproduction of cerebrospinal fluid is well-exemplified by the case of Vigoroux<sup>29</sup> whose patient had a fistula that communicated the ventricular system with the nose, resulting in the loss of cerebro-



spinal fluid which amounted to as much as 800 ml. of fluid per day. At the time of post mortem examination, this patient had a papilloma of the choroid plexus. Similar evidence was presented in the case of Ray, B. S. and Peck in 1956.<sup>19</sup>

The overproduction of cerebrospinal fluid is not necessarily the only way in which hydrocephalus might be produced in this condition. Since these tumors have a very rich vascular network within their connective tissue network, hemorrhage from them is not uncommon. A detached blood clot following such a hemorrhage may obstruct one or several of the ventricular vicissitudes and thus produce an obstructive hydrocephalus.

Another method by which hydrocephalus might be produced is by the obstruction of one or several of the foramina by the tumor itself. Such was the case in one other papillary tumor of the choroid plexus seen by us.

Dorothy Russell<sup>22</sup> cautions against diagnosing hypersecretory hydrocephalus in papillary tumors of the choroid plexus without extensive investigation for any degree of hemorrhage from the tumor itself might result in ependymitis or leptomenigitis or both or in gliogenous stenosis of the aqueduct and be followed by hydrocephalus.

Another type of obstructive hydrocephalus is that which occurs from the presence of a congenital obstruction of the aqueduct by a membrane, or by aqueductal stenosis or forking, as exemplified by the reported case.

Again, one must be careful in the evaluation of such a membrane in the Sylvian aqueduct and the labelling of the same as congenital, for it may consist of nothing more than glial tissue and be a part of a granular ependymitis from the hydrocephalus itself. Similar membranes have been described by Laurence.<sup>13</sup>

Due to the manner in which these tumors mimic the normal choroid plexus, one might encounter difficulty in answering the academic question of where choroid plexus hyperplasia ends and true papillary tumor begins.

Although, as mentioned before, ventriculography is probably the most important tool in the diagnosis of papilloma of the choroid plexus, the often associated abnormal clinical characteristics of the cerebrospinal fluid might be of helpful diagnostic value; namely, increased cerebrospinal fluid pressure, presence of xanthochromic fluid or of frankly hemorrhagic fluid, or occasionally, as in the case of Musoelin in 1940,<sup>17</sup> the presence of desquamated tumor cells in the cerebrospinal fluid.

The papilloma of the choroid plexus, as mentioned before, has a malignant counterpart which is capable of both local invasiveness and distant metastasis, but seeding within the subarachnoid space must not be confused with metastasis. Histologically, the tumor might be perfectly benign looking and yet be able to produce seeding and growth of new tumors elsewhere in the ventricular system or in the subarachnoid spaces of brain and spinal cord. This is entirely analogous to the behavior often encountered in the papillary cystadenoma of the ovary.

Although calcifications within the choroid plexus are

not uncommon whether a tumor is present in this location or not, the presence of well-formed bone in this location is a rare occurrence as has been mentioned by Bonnet (1861),<sup>2</sup> Sachs and Whitney.<sup>23</sup>

In the case that we have presented, the factors that resulted in the production of the hydrocephalus are not only those of hypersecretion but also the mechanical factor of obstruction of the Sylvian aqueduct by the membrane, for although ventricular dilatation occurred both above and below the membrane, the disparity in the dilatation of the lateral ventricles versus that of the fourth ventricle was quite evident. Due to the ocular pigmentation noted in the clinical examination, toxoplasmosis had to be considered in the differential diagnosis. However, this pigmentation, although spectacular, unfortunately is not pathognomonic of toxoplasmosis and, as mentioned before, the X-ray pictures of the post mortem brain showed calcifications only within the choroid plexus.

Congenital infection of the infant with toxoplasma Gondii is almost always the result of maternal infection during late pregnancy, and a titer of 1/4 is one which is found in the majority of healthy adults. A baby with toxoplasmosis has always a titer of dye test, although the intracutaneous toxoplasmin test may be negative until the age of 6 to 8 months.

Cytomegalic inclusion disease was also considered in the differential diagnosis, militating against it are the negative urine cultures for virus and the absence of cytomegalic inclusions in any of the other organs, particularly in the parotid gland which is always involved in this disease.

Papillary tumors, therefore, should be considered whenever hydrocephalus is encountered, especially in children.

Ventriculography is indicated in the differential diagnosis of hydrocephalus, although Laurence et al, 1961<sup>13</sup> caution that the latter procedure should be more than the usually employed bubble ventriculogram, and they suggest the replacement of a large volume of cerebrospinal fluid by air.

The importance of early diagnosis and treatment of this condition cannot be stressed enough, for, obviously, the sooner that treatment is instituted, the better the prognosis will be.

#### SUMMARY

A rare case of bilateral papilloma of the choroid plexus in a five-and-a-half-year old hydrocephalic girl is presented.

The tumor encountered at autopsy was not suspected during life, the existing hydrocephalus being attributed to either cytomegalic inclusion disease or toxoplasmosis.

After post mortem examination, it was evident that the hydrocephalus was due to two factors; hypersecretion from the tumor itself, and obstruction at the level of the Sylvian aqueduct by a delicate, tense, translucent membrane.



An interesting and uncommon additional feature of this case was the presence, within the choroidal papilloma, of well-formed Haversian bone.

A plea is made for regularly considering papilloma of the choroid plexus in the differential diagnosis of hydrocephalus in children, for here, early diagnosis and treatment might conceivably result in total cure.

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#### BRIGHT FUTURE IN MENTAL DISEASE PREVENTION

The keys to the rapid progress we are making in the management of emotional disturbances are the new drugs and the treatment techniques which are at our disposal. We now measure treatment in terms of days, weeks, or months instead of years and decades. These medical advances also mean that the psychiatrist no longer stands alone — all physicians, regardless of specialty, have tools and knowledge enough to diagnose and manage many forms of mental illness. Nor have we yet really touched prevention's great potential. — Millard B. Bethel, M.D., in *Hawaii Medical Journal*, January-February 1964.



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## An Overview of the Nursing Home Field\*

HARRY ALLEN, M.D.\*\*

### FOREWORD

This presentation by Dr. Allen is of particular interest in view of the current concern of the State Medical Advisory Committee with the nursing home problem in Maine. James H. Bonney, M.D., of Portland is chairman of a special sub-committee of five from the State Committee and is directing a study of the medical, social and rehabilitative needs of public assistance patients in nursing homes. The assistance of the Public Health Service, particularly that of Dr. Allen and his staff, is proving most helpful in the nursing home care study project—Dean Fisher, M.D., Commissioner.

The past 25 years have been firmly established in history for their tremendous scientific achievements. The entire field of medical care is in a state of flux as a result of this dramatic advance of medical technology and social change. My appearance before this group, is, in itself, indicative of this change, since the interest of public health in developing comprehensive programs in the field of chronic diseases has been relatively recent.

Major technological advances have completely altered the common causes of death. The chronic diseases now figure predominately in today's obituaries. Diseases of the heart and circulatory system alone cause more than half of the deaths in the United States. Cancer, diabetes and arthritic conditions take their toll. However, many of these deaths can be considered terminal diseases in men and women who have had an opportunity to live out a full and relatively healthy life.

Scientific and medical progress has resulted in a greatly decreased death rate with a commensurate increase in life expectancy. This has resulted in an increasingly large proportion of our population in the 65 years and over age group. As would be expected the chronic diseases are the main cause of disability. The fact that we are living longer, as well as the resulting changing pattern of disease, is an important factor in the increasing demand for nursing home beds.

Other factors are:

1. The concept of the general hospital for acute illness,
2. Steadily increasing cost of hospitalization associated with the prolonged course of the chronic diseases,

3. Housing — the design of contemporary housing makes it difficult to care for chronically ill and aging relatives at home,

4. Health insurance coverage for the aging,

5. Social Security and the availability of a source of funds through public assistance programs.

The continually rising costs of caring for the chronically ill and aging have made this a personal as well as a *community problem* of great importance. The needs of the patients and their families call for the involvement of a multiplicity of agencies and programs and have brought the public health authorities into the realm of medical care. The care of patients in nursing homes and related facilities is now one of their major concerns.

The interest of public health in developing comprehensive programs in this field is relatively recent. Since we do not know how to prevent most chronic diseases, the primary goal must be to develop programs and *facilities* which will limit disability and result in the return to maximum activity. A whole new series of programs supporting the care of patients in the out-of-hospital environments has evolved as a direct result of this concept. These programs permit the discharge of *selected patients* for follow-up in the home. These supportive programs help the patient live as normal a life as possible and include: coordinated home care; nursing care of the sick at home; homemaker services; meals on wheels; friendly visitors and social services including information and referral services. However, it should be realized that this is no panacea and that provision of these services is also costly.

The demand for increasing standards and the provision of a complete range of care, including physical therapy, occupational therapy and psychiatry, have far-reaching implications. These facilities have an important role in the current concepts of medical care; and there is no question but that *the nursing home is here to stay*.

\*A paper presented at the New England Hospital Assembly, Boston, Mass., March 26, 1964.

\*\*Regional Program Director for Chronic Diseases, Public Health Service, U.S. Dept. of Health, Education and Welfare, Boston Regional Office, Region 1, 120 Boylston St., Boston, Mass.



There is emerging a more exact picture of what the modern nursing home is. The nursing home should and must be "a place of dignity, understanding and care for the terminal patient, the massive hemiplegic, for the very old, the debilitated and senile as well as for those with less severe maladies." But more than this, it can be a bridge between the hospital and the home and, hopefully, the return of some individuals to community life.

The often-discussed "quality of care" is a complex factor to evaluate. It is my intention to review some of these factors, with the full realization that exception is the rule and that programs and regulations vary greatly from State to State.

#### COMPREHENSIVE COMMUNITY CARE

The term "comprehensive community care" is employed to describe the inter-relationship and utilization of medical care facilities. In this concept the general hospital is the hub of all care which radiates to and from it. Increasing numbers of chronically ill and aged whose illnesses have been diagnosed and treatment begun in the hospital are transferred to nursing homes and related facilities. The responsibility of the nursing home is to continue the prescribed treatment and to lead the patient toward self-care in order to restore him to the fullest possible degree of function. The nursing home must have two doors. Perhaps the greatest weakness is the failure to include the nursing home as a medical care facility.

The basic data summarized in this paper are a part of a study completed in Kansas in 1959.<sup>1</sup> This study is being presented because of familiarity with it; and secondly, it is easier to be objective about someone else's situation. However, comparisons will be made with New England studies which are of prime concern to this audience. Of special interest is the positive correlation of data from two different geographic areas.

#### STUDY METHOD

The immediate objective of the Kansas study was to learn something about the social, economic and medical characteristics of the patients in Kansas homes with the development of programs to improve patient care as the ultimate goal. Information for the survey was obtained through a random sampling of 100 licensed nursing homes, adult boarding homes, and homes for the aged. Public health nurses interviewed the administrators and reviewed the patient records at the time of evaluation or relicensing of the homes. The resulting sample represented approximately one-fourth of the licensed homes in that State.

#### SIZE OF HOMES

As in the rest of the nation, there is a predominance of small homes. The homes surveyed ranged from a minimum of 3 patients to a maximum of 79. One-third (33%) had fewer than 10 patients; 90%, fewer than 25.

The 1962 Smillie-Curran Report<sup>2</sup> indicated 20% of the Maine nursing homes licensed for 5 beds or less. The median in this study was 14 beds. The Boston College Massachusetts Survey<sup>3</sup> (1963) found over 36% under 20 beds and 64% under 30 beds. It is difficult to provide adequate nursing care under 10 beds. *A minimum of 30 beds to a maximum of 60 beds have been stated as the optimal size of a nursing home* for efficiency, economy and adequate standard of care. The poor economic return will gradually force the small operators out of business.

#### TYPE OF OPERATION AND STAFF

Of the 100 homes included in the Kansas survey, 94% were proprietary. This, again, compares favorably with the Smillie-Curran study which found 97% of the homes operating as a profit-making enterprise.

Few proprietors have special training in nursing care of the chronically ill and aging. However, some homes are operated by physicians and nurses. *The type of administration cannot necessarily be equated with quality of care.*

Only six registered nurses and one practical nurse were employed in the 100 homes. (Those having a registered nurse "on call" were not included in the data.) The majority did not provide a staff physician. This staffing existed in a situation where the great majority of the homes were caring for sick and infirm patients. Every nursing home should have 24-hour coverage by registered nurses or licensed practical nurses, working under the direction of physicians licensed to practise in that State. The common answer to queries about patients' medical care is: "they have their own private physicians." This, again, is a poor measure of the quality of care and frequency of visits.

#### AGE DISTRIBUTION

For the nursing home population, the females outnumber the males and are usually older. More than two-thirds (67%) of the residents are more than 75 years of age. This compares favorably with Maine's population of 81.7% over 75 years, and the Boston College study with a median age of 80 years, and 74.4% over 75 years. One thing is certain, the advanced age and infirmity are limiting factors to the rehabilitation potential of these patients.

#### ECONOMIC STATUS

The economic status of the residents is also a factor in the quality of care available to them. Sixty-nine percent of the patients in the Kansas data were receiving public assistance as the only source of payment for their care. It is interesting to note that more females than males are private patients. This is probably due, among other factors, to the increased longevity of the females who outlive their spouses and receive insurance and other financial income. In the Boston College study the number on public assistance was approximately 71%. Over one-

half the patients in nursing homes in Maine are State welfare clients, and approximately 3.25 million dollars are expended on their care. This is big business!

#### MEDICAL CONDITION AND DISABILITY

Eighty-three percent of the patients are ambulatory to varying degrees, and 17% are bedfast.

Eighty percent of the patients in the Kansas Survey were completely continent of urine and feces. Another 20% had either partial or no control of bladder and bowels. Nineteen percent of 3,108 patients in Massachusetts were completely incontinent; and another 3%, at night only.

#### DIAGNOSTIC CONDITIONS

The admitting diagnosis was taken from the patient's medical record by the public health nurses who participated in the Kansas study. These data are of value since they provide a good index of the amount of skilled nursing care needed by the patients. However, the admitting diagnosis does not explain why the patient remains in the home months and even years after the original episode.

The length of stay of 2,990 patients in Massachusetts homes ranged from 3 days to 32 years. Over one-third remained less than one year. Were these transferred to another home or hospital? Did they expire; or did they return to activities within the community?

The most frequent conditions reported are the degenerative and chronic diseases to be expected in a population of this age; and it is in line with the most common diseases in the general population. Particularly significant is the fact that disorders of the brain and senility appear most frequently in the Kansas figures or data and account for approximately one-third of the major diagnoses.

Cerebral vascular accidents (strokes) totaled 5%. It is reasonable to assume that this figure should be higher because many patients having non-specified paralysis and hemiplegia were undoubtedly stroke victims.

One thing can be stated with conviction — the medical conditions of the patients in the Kansas study indicate a need for close medical supervision and the skilled nursing care best provided by a doctor, a registered nurse, or a licensed practical nurse.

#### WHERE DO WE GO FROM HERE?

The situation that confronts us is the large number of homes of small size and inadequate staff coupled with the increasing demands for beds and improved

quality of care. Education will be required to upgrade many homes. This can be provided by:

1. State health authorities providing education by inspection as well as technical program assistance.
2. Nursing Home-Hospital Affiliations — though loudly acclaimed, not yet wholly accepted or implemented.
3. Special Public Health Service programs such as:
  - a. Nursing Home Cost Study
  - b. Standard Accounting Procedures
  - c. Team Approach to Patient Care
  - d. Environmental Health Factors in Nursing Homes, a 21-volume Syllabus
  - e. Regional and State Conferences
  - f. Nursing Home Standards Guide, a 64-page booklet published in 1963

Consultation to nursing homes is available from the many specialists in Federal, State and local agencies involved in modern medical care. However, special emphasis should be placed on benefits accruing to both hospitals and nursing homes where they become affiliated.

Another important area is uniform classification of homes with proper placement of patients. Information and referral programs are having much success in this area. The goal is to match the patients' needs with the facility. This will require education of the physicians, hospitals, welfare agencies and the general public.

Because of age and infirmity "rehabilitation" in its pure sense may be limited. The goal should be to have the patients perform at the maximum possible, and the institution of measures to limit disability.

Finally, the homes themselves must develop better cost accounting, in-service training and better record keeping. Adequate staff is costly, and homes must be developed with a bed capacity that can insure economy and efficiency. The nursing home must be reserved for patients who require skilled nursing care. All other patients should, and can, be cared for in homes for the aging and "boarding homes."

It is my hope that eventually the concept of "comprehensive community care" will prevail, and the community hospital will work in close conjunction with community-sponsored nursing homes.

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# Program . . .

111th Annual Session  
Maine Medical Association

Sunday - Monday - Tuesday

JUNE 14, 15, 16 — 1964

*The Samoset, Rockland, Maine*



## Program

Arranged by the Scientific Committee

ROBERT L. OHLER, M.D., Togus  
Chairman

ALBERT L. HUNTER, M.D., Camden

RICHARD P. LANEY, M.D., Skowhegan



Dr. Ohler

## Information

### Registration:

Registration throughout the session will be in the Lobby at The Samoset. Registration fee \$1.00.

Sunday June 14 — 9:00 A.M. to 5:30 P.M.

Monday June 15 — 9:00 A.M. to 6:00 P.M.

Tuesday June 16 — 9:00 A.M. to 4:30 P.M.

**Telephone:** The number at The Samoset is Rockland, 594-8411.

### Sponsors:

The speakers for the scientific programs are sponsored in part by grants from; Eli Lilly and Company State Medical Convention Program, The Merck Sharp & Dohme Postgraduate Program, and The Maine Heart Association, Inc.

### Visiting Delegates:

Introduction of Visiting Delegates will take place at meetings of the House of Delegates on Sunday, June 14 or at the General Assembly, Monday afternoon, June 15.

### Technical Exhibits:

The forty-two companies who contribute to the program by their participation in the Technical Exhibit are listed on pages 111 through 112. Please show your appreciation for their support of the Maine Medical Association by visiting each of these exhibits. You can't miss them as you pass through the lobby enroute to the Ballroom (where the Scientific Sessions will be held) to the Dining Room or to the Golf Course.

### Special Exhibits:

These exhibits, which are listed on page 112, will be located in the Ballroom.

## Sunday, June 14

9:30 A.M. First Meeting of the House of Delegates

Call to order: THOMAS A. MARTIN, M.D., President-Elect

Speaker of the House: LINUS J. STITHAM, M.D.

12:30 P.M. Luncheon

3:00 P.M. Second Meeting of the House of Delegates

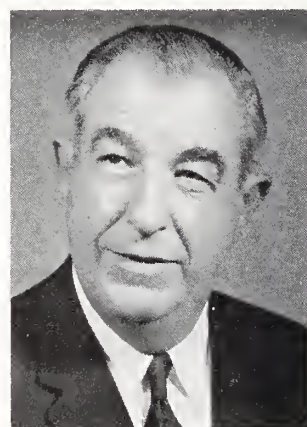
5:00 P.M. Visit the Technical Exhibits

6:30 P.M. Dinner

Speaker: DR. R. C. S. YOUNG, Birmingham, Michigan. Dr. Young appears through the courtesy of General Motors

Subject: **Meeting the Challenge of Leadership**

A lecturer throughout the United States and Canada, Dr. Young directs his efforts to rebuilding the climate for the American way of life.



Dr. Young

## Monday, June 15

### Scientific Program

#### Morning Session

*Presiding*—ROBERT L. OHLER, M.D.

9:30 A.M. **Frozen Blood**

CHARLES HUGGINS, M.D., Instructor in Surgery, Massachusetts General Hospital, Boston, Massachusetts

Sponsored in part by a grant from The Maine Heart Association, Inc.





Dr. Huggins



Dr. Hungate

10:15 A.M. to 12:00 NOON

**Emergency Medical Treatment of Disaster Victims.** Sponsored by the Maine Society of Internal Medicine and Medical Specialty Groups

10:15 A.M. **Medical Management of Disaster Victims during the Kansas City floods and after the tornado**

CARROLL P. HUNGATE, M.D., Kansas City, Missouri



Dr. Konecki



Mr. Reiman

11:00 A.M. **Panel: Medical Planning in Advance for Disaster**

Moderator, CARROLL P. HUNGATE, M.D.

**Hospital Staff Planning and Organization**

JOHN T. KONECKI, M.D., Radiologist, Lewiston

**Hospital Administration Preparation for Disaster Casualties**

PHILIP K. REIMAN, Administrator, Maine Medical Center, Portland

**Coordination of Medical Staff and Hospital Administration Plans and of Inter-Hospital Planning**

CHARLES W. STEELE, M.D., Internist and Cardiologist, Lewiston

11:35 A.M. Question and Answer Period

11:55 A.M. CARROLL P. HUNGATE, M.D. — Closing Summary

12:00 NOON to 2:00 P.M. Luncheon



Dr. Steele

**Scientific Program**

**Afternoon Session**

*Presiding*—ALBERT L. HUNTER, M.D.

**Sponsored by the Maine Chapter, American College of Surgeons**

1:30 P.M. Business Meeting, Maine Chapter, American College of Surgeons

2:00 P.M. **Hypovolemic Shock** (Including discussion of low molecular dextran and review of some Swedish experiences)

GEORGE R. DUNLOP, M.D., Chief of Surgery, The Memorial Hospital, Worcester, Massachusetts



Dr. Dunlop

3:00 P.M. **The Emergency Treatment of Disaster Victims With Emphasis Placed on Therapy of Open Wounds and Other Major Traumatic Injuries**

CARROLL P. HUNGATE, M.D., Kansas City, Missouri

4:00 P.M. **General Assembly**

*Presiding*, ERNEST W. STEIN, M.D., President

Election of President-Elect



Governor Reed

6:00 to 7:00 P.M. Social Hour, Dutch Treat, Ballroom

7:00 P.M. Annual Banquet

GOVERNOR JOHN H. REED, Guest of Honor

Presentation of Honorary Pins

Speaker: DR. JOHN C. KRANTZ, JR., Professor and Head of Pharmacology, University of Maryland School of Medicine, Baltimore, Maryland

Subject: **The Simplicity to Wonder**



Dr. Krantz

Dr. Krantz has written a book on public speaking in conjunction with Theodore McKeldin, the former Governor of Maryland, and his lectures are referred to by some of the medical students as "The Hour of Charm."

## Tuesday, June 16

### Scientific Program

#### Morning Session

*Presiding*—RICHARD P. LANEY, M.D.

9:30 A.M. **Diagnosis and Management of Sigmoid Diverticulitis**

ALFRED HURWITZ, M.D., Portland

10:00 A.M. **Nutritional Regulation of Plasma Lipids and Lipid Metabolism**

LAURANCE W. KINSELL, M.D., Director, The Institute For Metabolic Research, Highland-Alameda County Hospital, Oakland, California

Sponsored in part by a grant from The Merck Sharp & Dohme Postgraduate Program.



Dr. Hurwitz



Dr. Kinsell

11:00 A.M. **Pituitary Ablation in the Treatment of Diabetic Retinopathy**

CHARLES A. FAGER, M.D., Lahey Clinic, Boston, Massachusetts

12:00 NOON to 2:00 P.M. Luncheon



Dr. Fager



Dr. Adelson

### Scientific Program

#### Afternoon Session

*Presiding*—ROBERT L. OHLER, M.D.

Sponsored by the Maine Medico-Legal Society

2:30 P.M. **The Unwitnessed Homicide**

LESTER ADELSON, M.D., Pathologist and Chief Deputy Coroner, County of Cuyahoga, Cleveland, Ohio

6:30 P.M. Clam Bake

Presentation of Golf Prizes by  
DANIEL R. SHIELDS, M.D., Lewiston, Chairman  
Golf Tournament



## Specialty Group Meetings

### Monday, June 15

2:00 to 4:00 P.M.

#### Section on Ophthalmology

PAYSON B. JACOBSON, M.D., Portland, presiding

#### Management of Strabismus

ABRAHAM POLLEN, M.D., Associate Surgeon, Massachusetts Eye and Ear Infirmary; Assistant Professor, Ophthalmology, Tufts Medical School and Instructor in Ophthalmology, Harvard Medical School



Dr. Pollen



Dr. Tyson

#### Maine Society of Clinical Hypnosis

LEON R. JELLERSON, M.D., Boston, presiding

#### Physiology, Psychology and Hypnosis

DUDLEY B. TYSON, M.D., Anesthesiologist, Attleboro, Massachusetts

#### Maine Society of Obstetrics and Gynecology

EUGENE C. MCCANN, M.D., Portland, presiding

#### Recent Advances In Chemotherapy Of Gynecologic Cancer

HENRY M. WILLIAMS, M.D., Associate Attending Physician, Department Internal Medicine, Hartford Hospital; Assistant Clinical Professor of Medicine, Yale University School of Medicine and Clinical Assistant, Medical Neoplasia Service, Memorial-Sloane Kettering Cancer Center

#### Maine Society of Anesthesiology

ELIO BALDINI, M.D., Portland, presiding

#### Anesthesia for Emergency Surgery

PHILLIP S. MARCUS, M.D., Director, Department of Anesthesiology, Boston City Hospital

### Tuesday, June 16

10:00 A.M. Maine Medico-Legal Society

#### Business Meeting

IRVING I. GOODOF, M.D., Waterville, presiding

12:00 NOON Maine Radiological Society

#### Luncheon Meeting

FRANCIS J. O'CONNOR, M.D., Augusta, presiding

#### Video Tape Equipment for X-Ray

MR. JOHN E. TACKER, Vice President and New England Regional Manager, Picker X-Ray Corporation, Boston, Massachusetts



Mr. Tacker

12:00 NOON Maine Chapter, American Academy of General Practice

#### Luncheon Meeting

GEORGE W. BOSTWICK, M.D., Newcastle, presiding

2:00 to 4:00 P.M.

#### Maine Thoracic Society

DEAN FISHER, M.D., Augusta, presiding

#### Practical Aspects in the Management of Chronic Bronchitis, Asthma and Emphysema

SANFORD CHODOSH, M.D., Assistant Professor in Medicine, Tufts University School of Medicine; Research Associate, Lung Station, Boston City Hospital

#### Maine Society of Internal Medicine

CHARLES W. STEELE, M.D., Lewiston, presiding

#### Ear, Nose and Throat Group

JOHN E. WHITWORTH, M.D., Bangor, presiding

Maine Chapter of the American Academy of Pediatrics

RUSSELL A. MORISSETTE, M.D., Lewiston, presiding

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**Luncheon Meetings**

Monday, June 15

Advisory Committee to Secretary of State and to the Bureau of Motor Vehicles

Amy W. Pinkham Fund Committee

Tuesday, June 16

See Specialty Group Program

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**SPECIAL NOTICES**

**Election of President-Elect**

The Election of a President-Elect will take place at the General Assembly, June 15 at 4:00 P.M.

**Election of Councilors**

Election of Councilors for the following Districts will take place at the Second Meeting of the House of Delegates on Sunday, June 14 at 3:00 P.M.

Fifth District — Hancock and Washington

Sixth District — Aroostook, Penobscot and Piscataquis

In accordance with the By-Laws, "Nominations for members of the Council for any District where there is a vacancy shall be made by a caucus of the members of the House of Delegates of that District. Each candidate for Councilor must be a resident of the District for which he is nominated."

**Council Meetings**

The Council will meet on Saturday, June 13 and daily throughout the session at a time and place to be announced.

**Maine Chapter,  
American Academy of General Practice**

Social Hour

Sunday, June 14, 5:30 to 7:00 P.M.

**Tufts Medical Alumni Reunion**

Monday, June 15, 5:30 to 7:30 P.M.

**Golf Tournament**

DANIEL R. SHIELDS, M.D., Chairman

**HONORARY PINS**

Presentation of the Association's Honorary Pins will be made by Ernest W. Stein, M.D., President of the M.M.A., at the Annual Banquet, Monday evening, June 15 at 7:00 P.M.

**FIFTY-YEAR PINS**

Fifty-Year Lapel Pins will be presented to the following members who were graduated from Medical School in 1914:

**Androscoggin County**

Hudson R. Miller, M.D., Auburn  
Bowdoin Medical School

James A. Williams, M.D., Mechanic Falls  
Bowdoin Medical School

**Cumberland County**

C. Eugene Fogg, M.D., Portland  
Bowdoin Medical School

Thomas A. Foster, M.D., Portland  
Harvard Medical School

**Kennebec County**

Frederick T. Hill, M.D., Waterville  
Harvard Medical School

**Penobscot County**

Herbert C. Scribner, M.D., Bangor  
Bowdoin Medical School

**Piscataquis County**

Harvey C. Bundy, M.D., Milo  
University of Vermont College of Medicine

**York County**

Stephen A. Cobb, M.D., Sanford  
Harvard Medical School

**FIFTY-FIVE-YEAR PINS**

Fifty-Five-Year Pins will be presented to the following members who received Fifty-Year Pins in 1959:

**Cumberland County**

Joseph R. Ridlon, M.D., Gorham  
Bowdoin Medical School (1906)

**Penobscot County**

George I. Higgins, M.D., Newport  
Bowdoin Medical School

**York County**

Willard H. Bunker, M.D., York Harbor  
Bowdoin Medical School



**SIXTY-YEAR PINS**

Sixty-Year Pins will be presented to the following members who received their Fifty-Year Pins in 1954:

**Cumberland County**

Charles L. Cragin, M.D., Portland  
Bowdoin Medical School

**Lincoln-Sagadahoc County**

Harry F. Morin, M.D., Bath  
Boston University School of Medicine

**Penobscot County**

Carl J. Hedin, M.D., Brewer  
Dartmouth Medical School

**Piscataquis County**

Wilbur E. MacDougall, M.D., Bangor  
College of Physicians and Surgeons of Baltimore

**York County**

James W. Sever, M.D., Cambridge, Massachusetts  
Harvard Medical School (1901)

Ray L. Whitney, M.D., Cape Porpoise  
Harvard Medical School

**Visiting Delegates**

The Connecticut State Medical Society  
CHARLES M. BARBOUR, M.D., West Hartford  
SYDNEY LURIA, M.D., Bridgeport

The Massachusetts Medical Society  
EDWIN T. WYMAN, M.D., Boston

The New Brunswick Medical Society  
E. R. DAVIS, M.D., Saint John, N. B.

The Rhode Island Medical Society  
HANNIBAL HAMLIN, M.D., Providence

Vermont State Medical Society  
W. HERBERT JOHNSTON, M.D., Montpelier

**Delegates to Out-of-State Meetings**

The Connecticut State Medical Society  
ROBERT M. KNOWLES, M.D., Portland

The Massachusetts Medical Society  
HARLAND G. TURNER, M.D., Norridgewock

The New Brunswick Medical Society  
HAZEN C. MITCHELL, M.D., Calais

The New Hampshire Medical Society  
HOWARD P. SAWYER, JR., M.D., Portland

Medical Society of the State of New York  
ERNEST W. STEIN, M.D., Pittsfield

The Rhode Island Medical Society  
LEONARD G. MIRAGLIUOLO, M.D., Bangor

**County Delegates****FIRST DISTRICT****Cumberland County Medical Society**

*Delegates:* Stanley B. Sylvester, M.D., 1377 Washington Ave.,  
Portland, Secretary  
(2 years)

Robinson L. Bidwell, M.D., 31 Bramhall St., Portland

Donald P. Cole, M.D., 45 Deering St., Portland

John F. Gibbons, M.D., 22 Bramhall St., Portland

Charles R. Glassmire, M.D., 58 Deering St., Portland

(1 year)

Merle S. Bacastow, M.D., 22 Bramhall St., Portland

Louis G. Bove, M.D., 12 Deering St., Portland

Philip S. Fogg, Jr., M.D., 173 Pleasant Ave., Portland

Howard P. Sawyer, Jr., M.D., 22 Bramhall St., Portland

Philip P. Thompson, Jr., M.D., 131 Chadwick St., Portland

Maurice Van Lonkhuyzen, M.D., 131 State St., Portland

*Alternates*

(2 years)

Lloyd G. Davies, M.D., 78 Main St., Fryeburg

Stanley W. Kent, M.D., 42 Deering St., Portland

A. Dewey Richards, M.D., 11 Gage St., Bridgton

George F. Sager, M.D., 18 Bramhall St., Portland

(1 year)

Clifford W. Gates, M.D., 130 Main St., Gorham

Clement A. Hiebert, M.D., 18 Bramhall St., Portland

Stephen E. Monaghan, M.D., 157 Pine St., Portland

Hugh P. Robinson, M.D., 27 Deering St., Portland

Howard M. Sapiro, M.D., 171 State St., Portland  
William J. Tetreau, M.D., 144 Spring St., Portland

**York County Medical Society**

*Delegates:* Charles W. Kinghorn, M.D., 4 Wentworth St.,  
Kittery, Secretary

Carl E. Richards, M.D., 34 Winter St., Sanford

Roger J. P. Robert, M.D., 331 Main St., Saco

Robert F. Ficker, M.D., Maine St., Kennebunkport

*Alternates*

Melvin Bacon, M.D., 122 Main St., Sanford

Stephen A. Cobb, M.D., 34 Winter St., Sanford

Kenneth E. Leigh, M.D., Brixham Rd., York

**SECOND DISTRICT****Androscoggin County Medical Association**

*Delegates:* Donald L. Anderson, M.D., 369 Main St., Lewis-  
ton, Secretary

Morris E. Goldman, M.D., 185 Webster St., Lewiston (1 yr.)

Louis N. Fishman, M.D., 185 Webster St., Lewiston (1 yr.)

George B. O'Connell, M.D., 11 Lisbon St., Lewiston (2 yrs.)

Edward L. Reeves, M.D., 179 Sabattus St., Lewiston (3 yrs.)

*Alternates*

Robert D. Wakefield, M.D., St. Mary's General Hospital,  
Lewiston (1 yr.)

Charles A. Hannigan, M.D., 85 Goff St., Auburn (1 yr.)

Frederick B. Lidstone, M.D., 117 Goff St., Auburn (2 yrs.)  
Gerard L. Morin, M.D., 104 Ash St., Lewiston (3 yrs.)

#### Franklin County Medical Society

*Delegates:* Maynard B. Colley, M.D., 14 Main St., Farmington, Secretary  
Paul E. Floyd, M.D., 2 Middle St., Farmington

##### *Alternate*

Wallace H. Duffy, M.D., 100 Main St., Farmington

#### Oxford County Medical Society

*Delegates:* Albert P. Royal, Jr., M.D., 82 Maine Ave., Rumford, Secretary  
H. Richard Bean, M.D., 241 Main St., Norway (1 yr.)  
Peter B. Aucoin, M.D., 87 Congress St., Rumford (2 yrs.)

##### *Alternates*

James A. MacDougall, M.D., 303 Penobscot St., Rumford (1 yr.)  
Walter G. Dixon, M.D., 16 Deering St., Norway (2 yrs.)

### THIRD DISTRICT

#### Knox County Medical Society

*Delegates:* Onni C. Kangas, M.D., 417 Main St., Rockland, Secretary  
Harry G. Tounge, M.D., 12 Union St., Camden  
Johan Brouwer, M.D., 5 Beech St., Rockland

##### *Alternate*

William W. Ward, M.D., P. O. Box 804, Rockland

#### Lincoln-Sagadahoc County Medical Society

*Delegates:* George W. Bostwick, M.D., Newcastle, Secretary  
Ralph C. Powell, M.D., Damariscotta  
John F. Andrews, M.D., 20 West St., Boothbay Harbor

##### *Alternates*

Mary J. Tracy, M.D., Bristol Rd., Damariscotta  
Paul A. Fichtner, M.D., 781 High St., Bath

### FOURTH DISTRICT

#### Kennebec County Medical Association

*Delegates:* Earle M. Davis, M.D., 2 School St., Waterville, Secretary  
Brinton T. Darlington, M.D., Doctors Park, 89 Hospital St., Augusta  
John D. Denison, M.D., 105 Brunswick Ave., Gardiner  
Lane Giddings, M.D., 6 E. Chestnut St., Augusta  
Napoleon J. Gingras, M.D., 6 E. Chestnut St., Augusta  
Samson Fisher, M.D., 173 Main St., Waterville

##### *Alternates*

Oakley A. Melendy, M.D., Doctors Park, 89 Hospital St., Augusta  
William N. Runyon, M.D., 283 Water St., Augusta  
Richard E. Barron, M.D., Main St., Monmouth  
Irving I. Goodof, M.D., Thayer Hospital, Waterville  
Paul A. Jones, Jr., M.D., 2 School St., Waterville

#### Somerset County Medical Society

*Delegates:* Marian L. Strickland, M.D., Easy St., Canaan, Secretary  
George E. Sullivan, M.D., R.F.D. No. 1, Fairfield  
Harland G. Turner, M.D., Box 38, Norridgewock

##### *Alternates*

Edgar J. Smith, M.D., 1 Park St., Fairfield  
John P. Dow, M.D., 59 Main St., Pittsfield

#### Waldo County Medical Society

*Delegates:* Seth H. Read, M.D., 15 Church St., Belfast, Secretary  
Norman E. Cobb, M.D., 132 Main St., Belfast

##### *Alternate*

George L. Temple, M.D., Fahey St., Belfast

### FIFTH DISTRICT

#### Hancock County Medical Society

*Delegates:* Russell G. Williamson, M.D., Blue Hill Memorial Hospital, Blue Hill, Secretary  
Russell M. Lane, M.D., Water St., Blue Hill  
Llewellyn W. Cooper, M.D., 194 Main St., Bar Harbor

##### *Alternates*

Herbert T. Wilbur, Jr., M.D., 100 Main St., Southwest Harbor  
Philip L. Gray, M.D., Blue Hill

#### Washington County Medical Society

*Delegates:* Karl V. Larson, M.D., East Machias, Secretary  
James C. Bates, M.D., Eastport

##### *Alternate*

Hazen C. Mitchell, M.D., Calais

### SIXTH DISTRICT

#### Aroostook County Medical Society

*Delegates:* Clyde I. Swett, M.D., 18 Sherman St., Island Falls, Secretary  
Raymond G. Giberson, M.D., 156A Academy St., Presque Isle (3 yrs.)  
R. Paul Johnson, M.D., Main St., Fort Kent (2 yrs.)  
John B. Madigan, M.D., Houlton (1 yr.)

##### *Alternates*

H. Douglas Collins, M.D., Caribou Clinic, Caribou (3 yrs.)  
Melvin R. Aungst, M.D., Morneault Bldg., Fort Kent (2 yrs.)  
George J. Harrison, M.D., Market Sq., Houlton (1 yr.)

#### Penobscot County Medical Association

*Delegates:* Hadley Parrot, M.D., 74 Somerset St., Bangor, Secretary  
Lloyd Brown, M.D., 186 State St., Bangor  
Leonard G. Miragliuolo, M.D., 10 Maple St., Bangor  
George W. Wood, III, M.D., 156 No. Main St., Brewer  
Carl E. Blaisdell, M.D., 47 Broadway, Bangor  
Arthur N. Lieberman, M.D., 180 Broadway, Bangor

##### *Alternates*

Irvin E. Hamlin, M.D., Main St., East Millinocket  
Charles D. McEvoy, Jr., M.D., 186 State St., Bangor  
Clement S. Dwyer, M.D., 205 French St., Bangor  
George O. Chase, M.D., 489 State St., Bangor  
William M. Shubert, M.D., 317 State St., Bangor

#### Piscataquis County Medical Society

*Delegates:* Odd S. Nielsen, M.D., 85 Pleasant St., Dexter, Secretary  
Linus J. Stitham, M.D., 50 Main St., Dover-Foxcroft

##### *Alternate*

Charles H. Lightbody, M.D., No. Main St., Guilford



## Technical Exhibits

- Abbott Laboratories, North Chicago, Illinois**  
Representatives: Mr. A. J. Mack, Mr. A. Tancredi, Mr. W. A. Towne and Mr. P. F. Woodlock
- The Alkalol Company, Taunton, Massachusetts**  
Representative: Mr. Edward W. LeClair
- Ayerst Laboratories, 245 Paterson Ave., Little Falls, New Jersey**  
Representative: Mr. Ed McMahon
- Bicknell Photo Service, 24 Free St., Portland, Maine**  
Representatives: Mr. G. Roger Baker, Mr. Fred Wormell and Mr. John Walker
- Elmer N. Blackwell, Surgical Appliance Specialist, 565 Congress St., Room 207, Portland, Maine**  
Representatives: Mr. Elmer N. Blackwell and Mr. Oakley R. Sanborn
- The Borden Company, 350 Madison Ave., New York 17, New York**  
Representatives: Mr. J. R. Galvin and Mr. R. S. Marr
- Brewer & Company Inc., 67 Union St., Worcester 8, Massachusetts**  
Representative: Mr. Walter Spaulding
- Burroughs Wellcome & Co. (U.S.A.) Inc., 1 Scarsdale Rd., Tuckahoe, New York**  
Representatives: Mr. R. Parke, Jr. and Mr. L. C. Gee
- Carnation Company, 5045 Wilshire Blvd., Los Angeles 36, California**  
Representatives: Mr. William L. Galatas, Mr. Robert L. Garofano, Mr. Russell B. Mundi and Mr. Roger W. Carey
- Ciba Pharmaceutical Company, 556 Morris Ave., Summit, New Jersey**  
Representatives: Mr. John H. Angis and Mr. John F. Sullivan
- The Coca-Cola Company, P. O. Drawer 1734, Atlanta 1, Georgia**
- Endo Laboratories, Inc., 100 Stewart Ave., Garden City, Long Island, New York, 11533**
- Geo. C. Frye Company, 685 Congress St., Portland, Maine**  
Representatives: Mr. John F. Kimball, Mr. Hubert A. Honan, Mr. Sidney F. Cheney, Mr. Robert S. Cheney and Mr. S. Bruce Randall
- Geigy Pharmaceuticals, P. O. Box 430, Yonkers, New York**  
Representatives: Mr. Vincent L. Carson and Mr. Fred Muster
- Lederle Laboratories, Pearl River, New York**  
Representatives: Mr. R. Maffei and Mr. R. Pelletier
- Maine Surgical Supply Co., 233 Vaughan St., Portland, Maine**  
Representatives: Mr. Robert Axelsen, Mr. Lawrence Gardiner, Mr. George H. Munroe, Mr. Louis Olore and Mr. Philip Dana, Jr.
- McNeil Laboratories, Inc., Camp Hill Rd., Fort Washington, Pennsylvania, 19034**  
Representative: Mr. Joseph A. Ruest
- Mead Johnson Laboratories, Evansville 21, Indiana**  
Representatives: Mr. Kendall Dow and Mr. George McLay
- Merck Sharp & Dohme, West Point, Pennsylvania**  
Representatives: Mr. William Haskell and Mr. Robert Baxendale
- The Wm. S. Merrell Company, Cincinnati 15, Ohio**  
Representative: Mr. Joseph F. Crozier
- Witold J. Mikelk, 11 Field St., Auburn, Massachusetts**
- The National Drug Company, 4663 Stenton Ave., Philadelphia 44, Pennsylvania**  
Representative: Mr. William P. Dunbar
- Parke, Davis & Company, Detroit 32, Michigan**  
Representatives: Mr. L. S. Bosworth and Mr. E. E. Giroux
- Pfizer Laboratories, 235 East 42nd St., New York 17, New York**
- Thomas W. Reed Company, 533 Commonwealth Ave., Boston 15, Massachusetts**  
Representative: Mr. Richard D. Lund
- Riker Laboratories, Inc., Northridge, California**  
Representatives: Mr. Louis H. Celentano and Mr. John J. Cella
- A. H. Robins Company, Inc., 1407 Cummings St., Richmond 20, Virginia**
- Roche Laboratories, Nutley 10, New Jersey**  
Representatives: Mr. Peter Davis and Mr. John Strangio
- J. B. Roerig and Company, 230 Brighton Rd., Clifton, New Jersey**  
Representative: Mr. Clarence J. Johnson
- William H. Rorer, Inc., 500 Virginia Dr., Fort Washington, Pennsylvania**  
Representatives: Mr. Jefferson Beward, Mr. Edward T. Croke and Mr. Robert Roffler
- Ross Laboratories, Box 1317, Columbus 16, Ohio**  
Representatives: Mr. Harold Hutchinson and Mr. Dick Kaufman
- Sandoz Pharmaceuticals, Hanover, New Jersey**  
Representative: Mr. Herman Emidy
- Schering Corporation, 1011 Morris Ave., Union, New Jersey**  
Representatives: Mr. Floyd Selby and Mr. Jack Arlaud
- Sealy Mattress Company, 38 Everett St., Allston, Massachusetts**  
Representative: Mr. Lynn Foss

**G. D. Searle & Co., P. O. Box 5110, Chicago, Illinois, 60680**

Representatives: Mr. J. H. Muncaster and Mr. A. L. Grimes

**Seltzer & Rydholm, Inc., 250 Minot Ave., Auburn, Maine**

Representative: Mrs. Mary Ann Caron

**Smith, Miller & Patch, Inc., 902 Broadway, New York 10, New York**

Representatives: Mr. Paul Woodward and Mr. Kenneth Mullen

**E. R. Squibb & Sons, 745 Fifth Ave., New York, New York, 10022**

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**The Stuart Company, 3360 East Foothill Blvd., Pasadena, California**

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**The Upjohn Company, 700 Portage Rd., Kalamazoo, Michigan, 49001**

Representatives: Mr. W. J. Kayatta, Mr. J. J. Maloney, Mr. K. D. Locke and Mr. P. G. Hudson

**Wallace Laboratories, Cranbury, New Jersey**

Representatives: Mr. Walter Dobrolet and Mr. Joseph Moriarty

**Warner-Chilcott Laboratories, Morris Plains, New Jersey**

Representative: Mr. Joseph Verrengia

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## Special Exhibits

**AMPAC and MEMPAC**

**Associated Hospital Service of Maine**

**Boston Medical Reports**

**Division of Cancer Control of the State of Maine  
Department of Health and Welfare**

**Division of Eye Care and Special Services of the  
State of Maine Department of Health and  
Welfare**

**Maine Cancer Society**

**Maine Chapter, American Academy of General  
Practice**

**Maine Heart Association**

**Maine Radiological Society**

**Maine Society of Clinical Hypnosis**

**Maine Society of Internal Medicine**

**Maine Society of Pathologists**

**Medic-Alert Foundation**

**New England Physicians Retirement Program**

**Rehabilitation**

**Veterans Administration Center — The Solitary  
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*Research in the Service of Medicine*

## *Maine Heart Association Notes*



### **Cigarette Smoking And The Role Of The Physician**

"A number of epidemiologic studies have demonstrated a substantial association between cigarette smoking and morbidity and mortality from coronary artery disease. . . . death rates from myocardial infarction in middle-aged men were found to be 50 to 200% higher among heavy cigarette smokers as compared with nonsmokers and pipe or cigar smokers. A few of these studies also offer evidence that morbidity and mortality from coronary artery disease decrease among those who have stopped smoking. . . .

"It can be reasonably projected that associated with cigarette smoking, there is a yearly increase of about 60,000 premature deaths from coronary artery disease among men 40 to 69 years of age in the United States. This is approximately equal to the estimated increase in deaths associated with cigarette smoking from all other diseases combined.

"Cigarette smoking has also been shown to have an adverse effect on peripheral vascular disease. . . .

"Epidemiologic, experimental, and clinical studies have demonstrated adverse relationships between cigarette smoking and lung cancer, chronic bronchitis, and emphysema. One of the complications of emphysema is cor pulmonale. . . .

"As indicated above, increased death rates for cigarette smokers are related to coronary artery disease, lung cancer, cerebrovascular disease, hypertension, peripheral vascular disease, emphysema, chronic bronchitis, certain other forms of cancer, and peptic ulcer. . . .

"Studies on the biologic and psychosocial nature of the smoking habit and on more effective methods of assisting those who wish to stop smoking are urgently needed."



## Necrologies

### CHARLES H. NEWCOMB, M.D.

1880–1964

Charles H. Newcomb, M.D., 83, of Clinton, Maine died on January 7, 1964. He was born in Newburgh, Maine on September 8, 1880, son of Augustus Adams and Elizabeth Neally Newcomb.

Dr. Newcomb attended Hampton Academy, Castine Normal School and received his medical degree from the Medical School of Maine in 1907. He did postgraduate study at the New York Eye and Ear Infirmary and the New York Postgraduate School in 1923.

He was assistant surgeon for the Canadian Pacific Railroad from 1907 to 1909 and then moved to Clinton in 1910 where he practiced until his death.

Dr. Newcomb was an Honorary member of the Maine Medical Association and the Kennebec County Medical Association, having received a 50-year pin in 1957 and a 55-year pin in 1962. He was also a member of the American Medical Association.

Surviving is his widow, Elizabeth Moore Newcomb.

### RICHARD N. GOLDMAN, M.D.

1930–1964

Richard N. Goldman, M.D., 33, died suddenly on February 8, 1964. He was born in Lewiston, Maine, son of Morris and Deborah Goldman on August 22, 1930. He was educated here in the elementary schools, graduating from Lewiston High School in 1948, and received his B.S. degree from Bates College in 1952. He graduated from Tufts University School of Medicine in 1956 following which he spent three years at the

Boston City Hospital as a surgical intern and surgical resident. He completed a three year urological residency at the Boston Veterans Administration Hospital in June 1962. During this time he was a teaching fellow at Boston University School of Medicine and Tufts University School of Medicine.

It should be remembered that he accomplished all of these goals despite the fact that he had been a diabetic since his youth. He completed his training program under the handicap of severe vascular complications, requiring surgery, related to his disease.

He was married to Bob Ann Granik on October 21, 1956 and had three children, two boys and a girl, ages six, four, and 15-months.

He returned to Lewiston in July 1962 to establish himself in the private practice of Urology. In this very short period of time he became endeared to us as a quiet, gentle, competent, sincere physician and a friend to all. His passing has left a deep nonreplaceable loss to his family, friends, and colleagues; yet better that we should live on having known him, admired him, and been inspired by him.

In Richard Goldman's passing, the will of God has prevailed, the wisdom of which we mortals find difficult to accept. Death and its finality must come to all of us on this earth, but with the comforting belief in our own individual faith that everlasting life endures.

In humble and loving recognition of the personal, professional, and living attainments of Richard Nissen Goldman

BE IT NOW RESOLVED, that the members of the Androscoggin County Medical Association express their deep sorrow for the untimely loss of our friend and colleague, and

That we point with reverent pride to his contributions to this community, and

That we extend our deep sympathies to his dear wife and children as well as to his parents, Dr. and Mrs. Morris Goldman.

JOHN A. JAMES, M.D.

## In Memoriam

### *Androscoggin County*

Richard N. Goldman, M.D. Lewiston

### *Aroostook County*

Romeo J. Levesque, M.D. Frenchville

### *Cumberland County*

Luther A. Brown, M.D. Portland

Hans V. Mautner, M.D. Brookline, Massachusetts

Charles H. Patton, Jr., M.D. Brunswick

Carl M. Robinson, M.D. Falmouth

Carol Schwartz, M.D. Portland

### *Hancock County*

Silas A. Coffin, M.D. Bar Harbor

### *Kennebec County*

Charles H. Newcomb, M.D. Clinton

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## County Society Notes

### KENNEBEC

A meeting of the Kennebec County Medical Association was held at the Hotel Cassini in Waterville, Maine on March 19, 1964. The President, Kenneth W. Sewall, M.D. called the meeting to order following a dinner.

Dr. Sewall read a communication from Peter Bowman, M.D., Chairman of the Maine Medical Association's Committee to Study the Problems of Long-Term Patient Care. Dr. Bowman requested information relative to providing satisfactory care of chronically ill patients in Kennebec county who do not require general hospitalization. He also asked for an expression of opinion regarding the role of physicians in planning facilities for chronically ill patients. Harold N. Willard, M.D., in charge of rehabilitation and chronic care at the Thayer Hospital, told of the increasing home care facilities in the Waterville area. Public health nursing, homemaker services, transportation facilities, and home nursing equipment are a few of the facilities which have been developed for the community by the chronic care program at the Thayer Hospital. Jean L. Bolduc, M.D., Director of the Sisters Hospital, stated that he felt the situation would be greatly improved when the new Seton Hospital is opened, leaving the physical plant of the old Sisters Hospital available to the Sisters of St. Joseph who will occupy the building and provide care for chronically ill patients. A committee will be formed by Presidential appointment for investigation of the care of the chronically ill patient in Kennebec county and will report back to Dr. Bowman's committee.

Manu Chatterjee, M.D. of Brunswick, Director of Clinical Research with the Cardiopulmonary Clinic at the Maine Medical Center, was introduced by Robert L. Ohler, M.D., Past President of the Maine Heart Association. The Maine Heart Association made Dr. Chatterjee's appearance possible. Dr. Chatterjee, whose subject was Some Medical Aspects of Open Heart Surgery, discussed the general problems met at the open heart surgical unit at the Maine Medical Center in maintaining tissue oxygenation by extra-corporeal blood perfusion as well as a few of the specific problems encountered and how they are averted.

George E. Sullivan, M.D. of Fairfield, Councilor for the Fourth District to the Maine Medical Association, noted that the number of advertisements in the Maine Medical Journal has decreased markedly and that the representatives of drug firms and other advertisers should be encouraged to place advertisements in the Journal.

The Kennebec County Medical Association held a meeting at the Veterans Administration Center in Togus, Maine on April 16, 1964. The meeting was called to order by the President, Kenneth W. Sewall, M.D. following a dinner.

Dr. Sewall appointed Dr. Harold N. Willard of Waterville, Chairman of a committee to investigate the care of the chronically ill patient in the county with Drs. John D. Denison of Gardiner and Brinton T. Darlington of Augusta as fellow committee members. This committee will report to Dr. Peter Bowman, Chairman of a similar committee for the Maine Medical Association.

Samson Fisher, M.D. reported from the M.M.A. House of Delegates Interim Meeting which was held on April 12 in Brunswick. Dr. Fisher made the observation that one county had previously listed the standing committees of the Maine Medical Association and requested that any members interested in serving on such committees make their interest known. Dr. Fisher felt that this should be done in Kennebec county and that the committees which resulted would be much more active and accomplish a great deal more. One of the items of business discussed at the House of Delegates was the negotiation of the upcoming Medicare contract. Dr. Sewall, who serves on the In-

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insurance Advisory Committee, asked that opinions concerning the Medicare contract should be communicated at once so that fair representation would be given.

Robert L. Ohler, M.D., representing Joseph S. Weltman, M.D., welcomed the Association to Togus and gave a brief report, as Chairman of the Scientific Committee of the Maine Medical Association, concerning the coming meeting of the Maine Medical Association in June.

Allan J. Stinchfield, M.D. introduced several orthopedists from throughout the State whom he had invited to attend the meeting in order to hear the speaker of the evening, Robert L. Preston, M.D., Clinical Professor of Orthopedic Surgery at the New York University Medical School. Dr. Preston spoke on Surgical Treatment of the Arthritic Hand which was followed by an interesting question and answer period.

EARLE M. DAVIS, M.D.  
*Secretary*

#### HANCOCK

The Hancock County Medical Society met at the Hancock House in Ellsworth, Maine on April 8, 1964.

The guest speaker was Harold A. Pooler, M.D., Superintendent of the Bangor State Hospital, who spoke on Psychiatry in General Practice. He presented his own problems in dealing with private physicians and reviewed the problems and complaints which private physicians present to him.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

#### CUMBERLAND

The regular monthly meeting of the Cumberland County Medical Society was held on April 16, 1964 at Boone's Restaurant, with 84 members and guests attending.

Following a social hour and turkey dinner, the meeting was called to order by the President, Charles R. Geer, M.D. Winton Briggs, M.D. was elected to membership in the society.

Franklin F. Ferguson, M.D. read a resolution on the death of Luther A. Brown, M.D. and G. Hermann Derry, M.D. added fitting comments. Boris A. Vanadzin, M.D. reported on the final plans of the Health Careers Committee for exposing high school students to the practice of medicine and he apologized for the unintended slight of the doctors located outside Portland for not being included in this year's plan. The society voted to hold an early fall meeting in Brunswick.

Mr. William M. Kitching of the U.S. Public Health Service discussed the increasing rate of gonorrhea in Maine and the threat of increasing incidence of syphilis. Dr. Vanadzin described the local problem and procedure for dealing with venereal disease.

Mr. Leon W. Stover and Mr. Richard M. Salisbury announced the program for promotion of Medic-Alert sponsored by the Southwestern Maine Life Insurance Underwriters and by the Maine Medical Association. The society voted to contribute to this program; the amount to be decided by the Executive Council of the Cumberland County Medical Society.

Philip P. Thompson, Jr., M.D. requested the sanction and support of the Cumberland County Medical Society in a survey of nursing homes in this area by the State Department of Health and Welfare and it was voted to approve this survey.

The secretary read a letter from the Office of the Surgeon General to Dr. Hanley requesting renegotiation of the Medicare contract for the State of Maine. After general discussion of the matter, with major comments by Drs. Maurice Van Lonkhuyzen and Albert Aranson, it was voted to support Dr. Aranson in his position as our representative on the Health Insurance Committee, knowing that his action would reflect the opinions of the members.

STANLEY B. SYLVESTER, M.D.  
*Secretary*

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## News, Notes and Announcements



Dr. Charles R. Geer (l), representing the Cumberland County Medical Society, presents a check supporting Operation Lifeguard to Richard Salisbury (c) of the Southern Maine Life Underwriters Association. Barbara Allen (r) of the WEEKDAY staff looks on.

#### WCSH-TV, Portland, Maine, Boosts Local Chapter Of Nationwide Health Organization

WEEKDAY ON SIX, WCSH-TV's daily public service feature program, recently aided in publicizing a nationwide effort to have individuals with chronic disorders, blood problems, etc., wear identifying bracelets. The Southern Maine Life Underwriters Association is promoting the program locally under the title, Operation Lifeguard.

Highpoint of the WEEKDAY ON SIX show was the presentation of a check from the Cumberland County Medical Society to Southern Maine Life Underwriters for the purpose of organizing and promoting Operation Lifeguard. People with special medical problems would submit their health history to a central agency in California and receive in turn a bracelet with special identifying numbers. Then, in case of accident, injury, etc., a doctor unfamiliar with the patient's health record would be able to quickly check by collect phone call with California and discover information vital in treatment.

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features the following new books in their full page advertisement appearing elsewhere in this issue:

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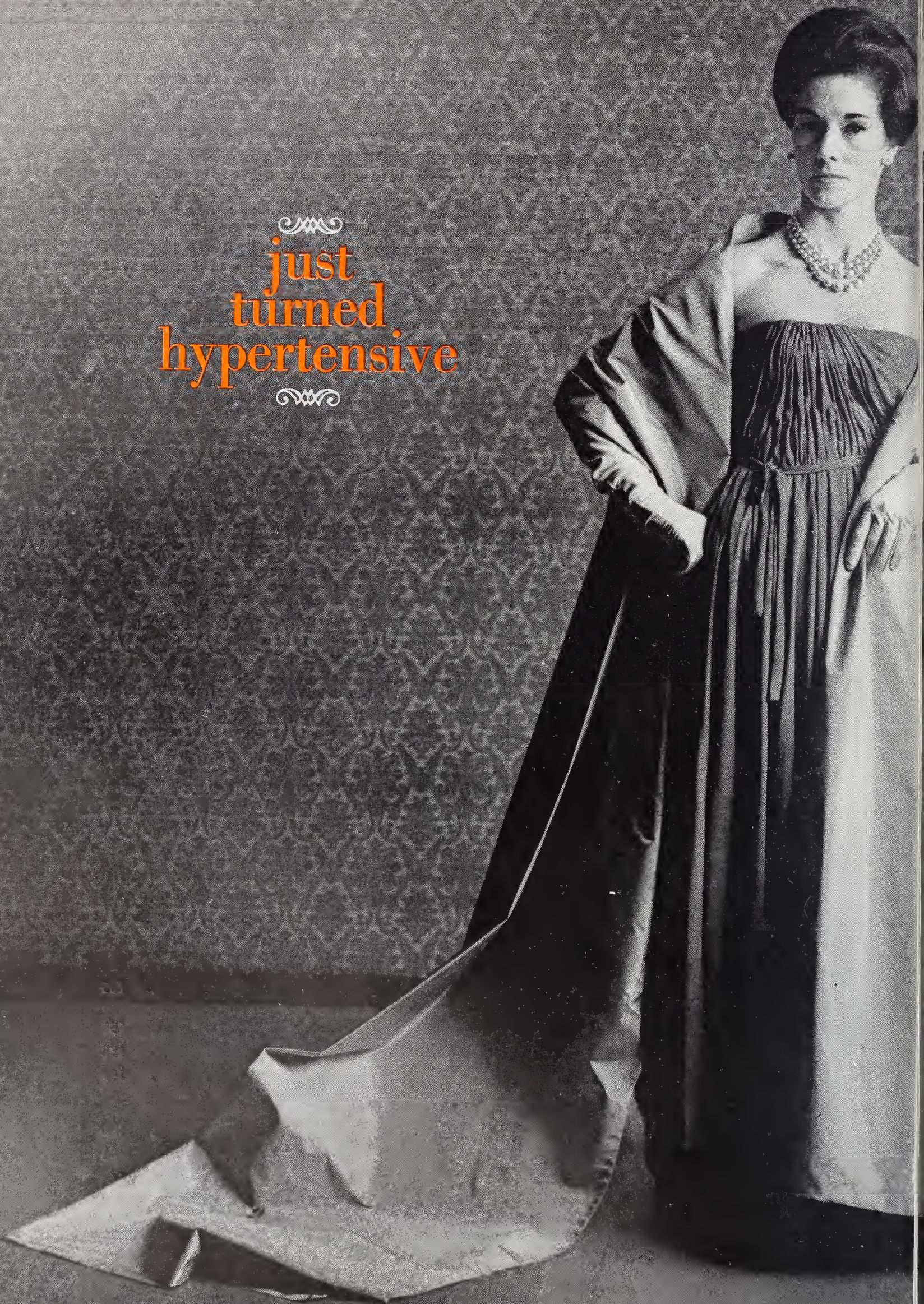
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# The Journal of the Maine Medical Association

Volume Fifty-Five

Brunswick, Maine, July, 1964

No. 7

## Excision of Pilonidal Sinus with Primary Closure A Ten-Year Study\*

FENNELL P. TURNER, M.D.

A technique of excision and primary closure of pilonidal sinus was published in the July, 1952, issue of the Journal of the Maine Medical Association.<sup>1</sup> As more than ten years have elapsed since the initial report, it may be of value to describe this procedure once again and present an analysis of both immediate and long-term postoperative results.

### CLINICAL MATERIAL

Two hundred and five patients with symptomatic pilonidal sinus were admitted to the hospital during the ten-year period covered in this study. One hundred and sixty-four patients were treated by excision, mobilization of musculofascial flaps, and closure with removable far-and-near sutures of stainless steel in accordance with the technique described below. Forty-one patients were treated by other methods (Table 1). Of those treated by excision and closure, 65 patients had one visible sinus tract, and 42 had two. In 57 patients three or more sinus tracts were found. In many of this latter group the sinus tracts were complicated and extensive. In 40 patients sinus openings were present lateral to the mid-line, and in 15 patients sinus ostia were present in the perianal region. Clinical evidences of acute infection were present on admission to the hospital in 52% of the 164 patients. In 19 of these, incision and drainage was carried out soon after admission to the hospital. Definitive curative surgery was then carried out in from one week to two months. In the 33 remaining patients conservative therapy consisting of warm applications and sitz baths was all that was required.

TABLE 1

SURGERY OF PILONIDAL SINUS Ten-Year Study in 205 Patients		<i>No. of Patients</i>
<i>Initial Operation</i>		
Excision and Primary Closure		164
Musculofascial flap operation		
(Incision and drainage pre-op. —	19)	
Miscellaneous Operations		41
Excision and simple primary closure	8	
Excision and partial closure	12	
Excision and packing	2	
Marsupialization	6	
Incision and drainage and/or cur- ettage (no subsequent surgical procedure this hospital)	13	
		<hr/> 205

In 28 of the 164 patients history was obtained of incision and drainage on one or more occasions prior to admission. In 36 patients definitive surgery had been previously carried out elsewhere, and in several of these repeated attempts to effect a surgical cure had been carried out.

### TECHNIQUE OF THE OPERATION

The lesion with all its ramifications is excised en bloc by means of a lengthwise elliptical incision, carried straight down to sacrococcygeal fascia. Removal of skin is kept to a minimum, and undermining of subcutaneous tissue is avoided. Additional wedge-shaped incisions are sometimes necessary to excise lateral tracts. Hemostasis is obtained by means of hot saline packs and electrocoagulation. Musculofascial flaps are developed in the manner described by Holman<sup>2</sup> (Fig. 1). The wound is then closed primarily with far-and-near pull-out wires of stainless steel, either double-0 multiple strands or 30-gage single strands (Fig.

\*From the Veterans Administration Center, Togus, Maine.



FIG. 1

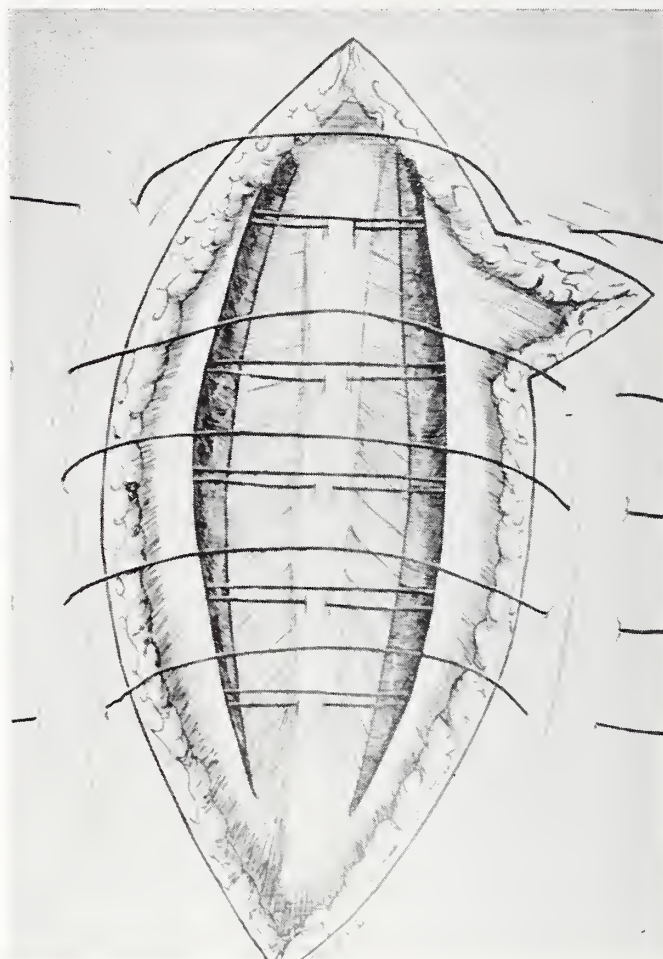


FIG. 2

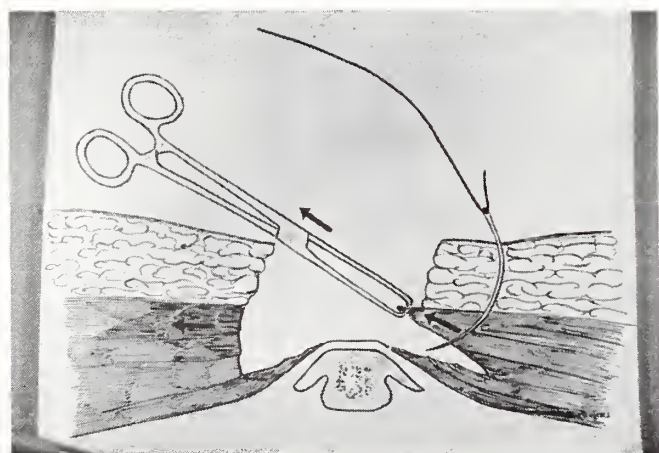


FIG. 3

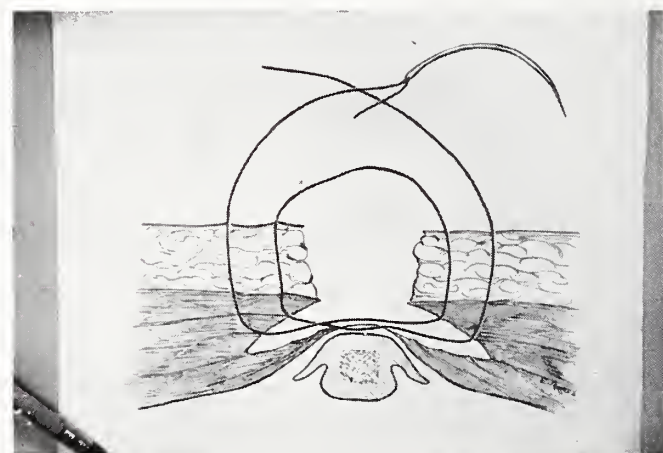


FIG. 4

2). Care is taken that the sutures are inserted through both skin and fascial layers at equal distances from the points of respective incision in each layer (Figs. 3 and 4). A bite is also taken in the sacrococcygeal fascia. With this type of suture the fascial layers are brought together in the midline almost as easily as subcutaneous tissues and skin, and all deadspace is effectively obliterated. Before closure the wound is irrigated with saline solution. Frequently if there is believed to be gross contamination, irrigation with antibiotic solution is carried out. The skin is then meticulously closed with vertical mattress sutures (Fig. 5), and the far-and-near sutures are then tied over two gauze rolls (Fig. 6). A separate strip of gauze is first drawn under the innermost wire loops, and a second gauze roll is then placed on top and under the second row of wire loops, which are then tied. Care is taken to express all fluid out of the wound before the gauze

rolls and the pressure dressings are applied. Before tying the wire sutures each one is carefully snugged up, first on one side of the wound and then on the other. Care is also taken that the adhesive straps are removed from the buttocks prior to tightening of the wire sutures. Following operation the patient is given clear liquids by mouth for several days, and bowel movements are thereafter prevented by means of low-residue diet and paregoric. The patient is ambulatory but is not permitted to sit down until after the wound has healed. All sutures are removed on the eighth or ninth postoperative day, and the patient is then ready for discharge from the hospital.

#### WOUND HEALING

Primary wound healing was obtained in 145 or 88.4% of the 164 patients. Wound hemorrhage, or



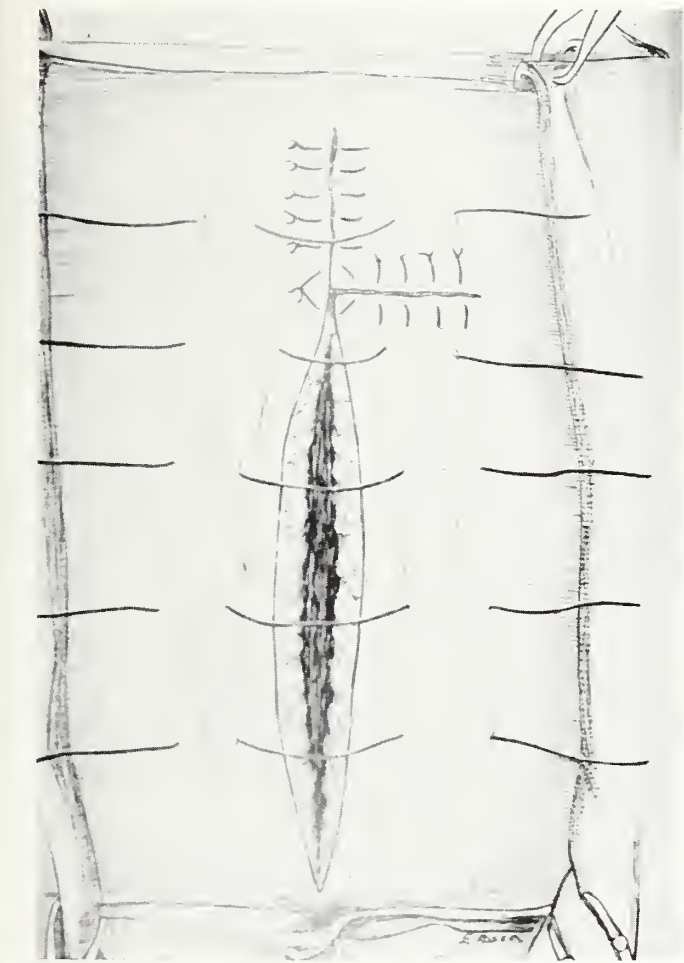


FIG. 5

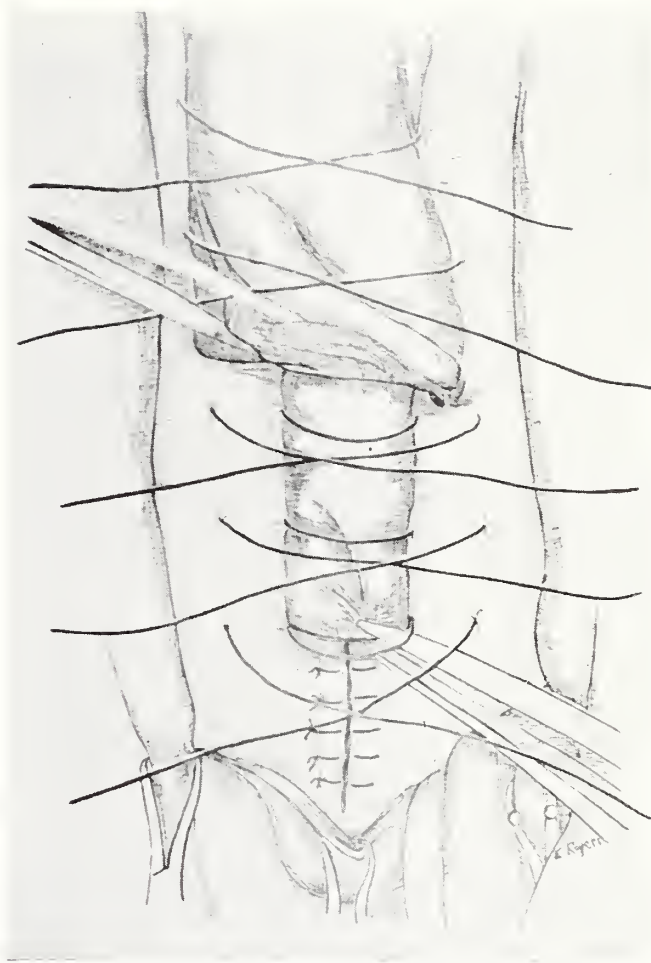


FIG. 6

TABLE 2

WOUND HEALING COMPLICATIONS FOLLOWING PRIMARY CLOSURE				
		No.	Percent	Healing Time
Major (Re-opening of wound required)	*Hematoma	10	6.1	3-10 wks. (failure to heal in 2)
	**Major infection	9	5.5	5-10 wks.
Minor (Reopening of wound not required)	Skin separation (small)	12	7.3	1-6 wks.
	Minor infection (wound not drained)	6	3.7	1-2 wks.
	*Retained wire fragments (technical error)	1	0.7	Failure to heal
		38	23.3	

\*Failure to heal — 3

\*\*Six of 9 major infections positive for Staphylococcus

hematoma, requiring evacuation occurred in 10 patients, or 6.1%; and major wound infection took place in 9, or 5.5% (Table 2). Minor complications not requiring reopening of the wound occurred in 19. In six patients out of this group of 19, seropurulent drainage was observed from one or more sutures. In each instance this responded to conservative therapy, and reopening of the wound was not required. In 12 of the 19, skin separations from 1 to 3 cm. in length took place. These minor complications were also treated conservatively and healed in from one to six weeks. In one patient where sutures had been removed outside the hospital, fragments of wire had been retained, resulting in a failure of wound healing.

RECURRENT PILONIDAL SINUS

Five patients have been lost to follow-up. Of the 159 patients followed, 143 were followed for more than one and one half years. There has been no dis-

TABLE 3

RECURRENT PILONIDAL SINUS	
Time of Recurrence	Number
0- 6 months	10
7-12 months	4
13-18 months	2
4 years	1
5 years	1
TOTAL	18

TABLE 4

RECURRENT PILONIDAL SINUS POSSIBLE FACTORS OF ETIOLOGICAL IMPORTANCE								
Name	No. Mid-Line Sinus Ostia	No. Lateral Tracts & Ostia	No. Perianal Tracts & Ostia	Prior Definitive Surgery	Prior I & D	Inflamma- tion on Admission	I & D This Admission	Wound Healing Complica- tions
L.T.	3	1				x		
H.K.	2			x		x		
H.C.	4			x	x			x
L.N.	1			x				
F.S.	1		1	x	x		x	
M.F.	1				x			
R.E.	Several			x				
R.G.	1				x			
H.R.	Several			x				x
R.B.	1			x		x		x
E.G.	2			x	x			
W.K.	4	1	1			x		
R.C.	1	1						
J.H.	2	2	2		x	x		x
J.L.	2		2		x	x	x	
R.S.	Several			x				x
C.F.	Several			x				
C.T.	2	1						

ability. Some of the patients complained of minimal tenderness in the early follow-up period, but this invariably disappeared with time. There were 18 known recurrences, and 16 of these recurred within 18 months after operation (Table 3). It is probably of significance that 10 of the 18 had previously had attempts at definitive surgical care elsewhere (Table 4), that seven others had had incision and drainage on one or more occasions prior to admission to the hospital and that seven patients were described on admission as having complicated, extensive disease with tracts extending out into either one or both buttocks or down to the perianal region. Six of the 18 also had evidences of acute infection at the time of admission to the hospital. In two of these incision and drainage had recently been carried out, and the wounds were unhealed at the time of definitive surgery. In five patients postoperative wound-healing complications had occurred, and in three of these five complete wound healing had failed to take place. Finally in 11 of the 18 more than one of the factors listed above were present. On the other hand, following the 74 primary-closure operations in uncomplicated cases of pilonidal sinus that were neither associated with recent acute infection nor in whom previous surgical therapy had been carried out there was only one recurrence (Table 5).

DISCUSSION

The musculofascial flap technique was first devised during the early years of World War II.<sup>3,4,5,6</sup> This operation was an improvement over simple excision and closure as it permitted sufficient mobilization of tissue so as to permit adequate wound closure without tension and with resulting effective obliteration of all dead-space. The technique described herein differs from

TABLE 5

RECURRENT PILONIDAL SINUS FOLLOWING PRIMARY CLOSURE Relationship to Clinical History in 164 Patients			
	No. of Patients	No. of Recur- rences	Per- cent
Uncomplicated, uninfected cases			
without previous surgical treatment	74	1	1.4
No previous surgical treatment	100	4	4.0
All cases with musculofascial flap operation	164	18	11.0
Patients with acute infection at time of admission to hospital	52	6	11.5
Patients with wound healing complications (major & minor) after flap operation	38	5*	13.2
History of I & D (only) on one or more occasions prior to admission	28	4	14.3
History of previous definitive surgical treatment on one or more occasions	36	10	27.8

\* Failure to heal following original operation in three out of these 5 patients.

others in the following details. There is no buried suture material, wound closure is obtained by means of removable far-and-near sutures of stainless-steel wire inserted through fascia and subcutaneous tissue as well as skin, and the wounds are closed without drainage. The far-and-near suture is not a commonly used stitch, but it is one that has been used for many years.<sup>7</sup> Babcock<sup>8</sup> has called it a combined relaxing and coapting suture, and he felt that it was one of the best suture techniques to use in the presence of wound tension. Excision and primary closure has several inherent advantages over the various open surgical techniques. Firstly, the period of postoperative hospitalization and/or morbidity is shortened. And secondly, there is no



necessity for daily dressings, which require frequent special visits to the hospital clinic or to the doctor's office. These factors have been important in our practice as many of the patients treated at the Veterans Administration Hospital come long distances for treatment, and many other patients live in relatively isolated rural districts, where medical care may be difficult to obtain. Proper postoperative care of such patients following any open operation would therefore require that such patients have prolonged hospitalization, and this of course is not necessary following a satisfactory procedure of excision and primary closure.

At first glance the musculofascial flap operation appears to be too radical and too extensive a procedure to carry out in the treatment of what seems to be such a minor lesion. However, it should be pointed out that there is little or no discomfort following operation, and the patient is ambulatory within a day or two after the operation. After removal of the sutures on the eighth or ninth day the patient is discharged from the hospital, and in general there is no necessity for further medical care. In the event that wound separation or wound hematoma occurs, secondary wound healing will take place within a reasonable length of time with conservative management. Even in the event that wound infection occurs, wound healing will not take any longer than following "open" methods of operation, where wound healing, regardless of the operative technique, generally takes from four to twelve weeks.

Following excision and primary closure and prior to discharge from the hospital it is important to give the patient careful instruction as to body hygiene. This is necessary in order to avoid ingrowing hairs or hair-follicle infection, which may be one of the important causes of recurrence in the primary-closure operation as it is also thought to be following the various open operations. Meticulous skin closure and avoidance of overlapping of the wound are also important in this regard. However, failure completely to remove diseased tissue is believed to be the commonest cause of recurrences. In several of our patients with recurrences, study of the excised specimen in the Laboratory had shown sinus tracts extending up to the margin of the cut specimen. It was the opinion of the pathologist in these cases that the disease had been incompletely removed. In one patient wire stay sutures had been improperly removed, and retained wire fragments later eroded through the skin, resulting in further sinus tracts. In other patients there was failure to heal following a hematoma or infection with subsequent evacuation. In some of the patients with recurrent disease excision and primary closure was again adopted as the treatment of choice (Table 6).

It is believed that postoperative wound infection can be held to a low level if care is taken not to carry out primary closure in the presence of severe infection (Table 7). As our wound infections have generally been due to antibiotic-resistant organisms, it has become evi-

TABLE 6

TREATMENT OF RECURRENCES FOLLOWING PRIMARY CLOSURE			
	No.	Healed	2nd Recurrence
Repeat Primary Closure	6	4	2
Open Operation	8	7	1
No Treatment desired	4	—	—
No. recurrences	18		

TABLE 7

WOUND INFECTION FOLLOWING PRIMARY CLOSURE Relationship to Pre-operative Evidence of Acute Infection			
	No. Pts.	No. Wnd. Inf.	Percent Wound Infection
No Evidence Acute Infection on Admission	112	3	2.7
Acute Infection on Admission (I & D Pre-op.)	52 (19)	12 (5)	23.1 (26.2)
All Cases	164	15	9.1

- Note:—
- 80% of all wound infections occurred in patients who had shown evidence of acute infection on admission to the hospital.
  - 6 of 9 major wound infections were due to resistant staphylococci.

dent that systemic antibiotic therapy is of little value in the operative management of these patients. We do not use systemic antibiotics following surgery for pilonidal sinus. We do irrigate the wound with saline solution, and on occasion we irrigate the wound with antibiotic solution if we think that contamination has taken place. It should be pointed out that six of the major infections which have taken place were due to antibiotic-resistant *Staphylococcus aureus*. Considerable judgment obviously must be used in determining the time of surgery in infected cases. In cases where the severity of the infection has required unroofing of the sinus tracts or perhaps actual incision and drainage, it is sometimes advisable to postpone plans for definitive surgery until evidence of recurrence and chronic disability become apparent. For one thing it is recognized that some patients following incision and drainage have no further trouble from this disease, presumably because of the destruction of the epithelial lining of the sinus by necrosis.

During this long study period a concerted attempt has been made to treat as many patients as possible by means of excision and primary closure. It was felt that only in this way could the true place of this operation in the surgical management of this controversial disease be accurately determined. As the 205 patients admitted to the hospital during this ten-year period were cared for by fifteen different surgeons, it has been only through the whole-hearted cooperation of the entire Surgical

Service that it has been possible to develop this long series, for this the author is extremely grateful. Nevertheless it should be pointed out that for the same reason it has been impossible to maintain a strict experimental approach through this ten-year period; and, as a result, for one reason or another, other surgical techniques have been occasionally used. Many of these patients were suitable for excision and primary closure when first seen, and others would have been suitable after prior treatment for acute infection. A retrospective analysis of these 41 patients added to the 164 treated by excision and closure would indicate that in the vicinity of 123 or 60% of all the patients admitted with pilonidal sinus could have been treated by excision and primary closure when first seen. Another 67, or 33%, could also have been so treated after prior treatment for acute infection. In only 15, or 7%, of all patients seen in this ten-year period can it be concluded that the disease process was too extensive to permit excision and primary closure.

Despite the feasibility of excision and primary closure as the definitive procedure for surgical cure in the majority of patients with pilonidal sinus, the question can now be asked, "Should they always be so treated?" To this we must answer, "No." Because of the high incidence of wound infection and the high recurrence rate in certain categories of patients it is probable that other techniques of treatment are occasionally to be preferred. Our current philosophy of management of this controversial lesion is therefore as follows:—

(1) In simple non-infected cases excision and primary closure is the operation of choice. Mobilization of musculofascial flaps may not always be necessary, particularly if the amount of tissue excised is small and if the location of the disease permits adequate wound closure.

(2) In more extensive disease and following a greater degree of tissue removal mobilization of musculofascial flaps is of value as it will permit a more thorough obliteration of deadspace and a more secure wound closure. In either type of primary closure it is important to restrict buried suture material to the minimum. Drainage is rarely necessary.

(3) Clear-cut, acute infection (33% of patients) must be adequately dealt with prior to definitive therapy. In the presence of frank abscess, incision and drainage is necessary. In lesser degrees of inflammation, marsupialization or incision and curettage may be used. In 3 to 6 weeks, if it appears that complete obliteration of the disease and sinuses is not forthcoming, excision and closure may then be carried out.

(4) In complicated pilonidal sinus disease, where extensive sacral, lateral, and/or perianal tracts are present (7% of patients seen with pilonidal sinus), unroofing of the tracts with marsupialization or other varieties of open methods of surgery will usually be required. In certain cases some areas may be excised and closed primarily and other areas of involvement (viz. multiple perianal ostia) may be left open. Following open operations frequent dressings are required in order to

TABLE 8  
RECURRENCE RATE FOLLOWING EXCISION OF PILONIDAL SINUS BY VARIOUS METHODS AS REPORTED IN THE LITERATURE

Type of Operation	Percent of Recurrence
Marsupialization	2 - 16
Open Operation (all types)	3 - 33
Primary Closure (all types)	7 - 32
Primary Closure (musculofascial flaps)	7 - 37

prevent the development of epithelial bridges, ingrowing nests of hair, or epithelialization of deep pockets.

SUMMARY

En bloc excision, mobilization of equal musculofascial flaps of gluteus maximus muscle, and primary closure by means of removable far-and-near sutures of stainless-steel wire have been carried out in 164 unselected patients with symptomatic pilonidal sinus. These patients were operated upon over a ten-year period of study by 15 different surgeons. Primary wound healing was obtained in 88% of the patients. Follow-up studies were carried out in 97% of the patients, and 81% of the patients were followed for more than 18 months. There were 18 recurrences or 11%. If technical errors could have been avoided and if better case selection had been carried out, it is believed that the recurrence rate could have been held down to less than 4%. This would compare favorably with the recurrence rates reported in the literature following various surgical procedures by different authors (Table 8). Recurrences were found to have been most frequent in patients who had previously had surgical treatment and in patients with extensive disease. This would lead us to believe that recurrence of pilonidal sinus usually is due to failure of excision of the diseased tissue and that in a few instances it is due to failure to obtain complete primary wound healing at the initial operation.

Excision and primary closure is always contraindicated in the presence of acute infection with abscess formation because of the increased risk of wound infection and because of the increased likelihood of recurrent disease in such cases. Relative contraindications for the primary-closure operation would include the following: persistent chronic infection due to resistant staphylococci; low-grade acute inflammation associated with erythema, induration, and purulent drainage; extensive secondary tracts either sacral, lateral or perianal; a history of definitive surgery on one or more previous occasions.

In conclusion, our enthusiasm for primary-closure technique of operative treatment of pilonidal sinus has continued to remain high. This operation in our experience has provided the patient with a thin, strong, non-tender scar with minimal deformity and a protective pad of fat and fascia over the bony prominences of the coccyx and sacrum. It is a simple operative procedure applicable to the majority of all patients admitted to the hospital with this disease. It can be carried out with a low morbidity and a reasonably low recurrence rate.



Wound infection can be held down to a level acceptable for any clean operative procedure so long as satisfactory clinical judgment is exercised as to the time of surgery and so long as the surgeon is careful to obtain complete hemostasis and a meticulous wound closure. It is our firm belief that this particular technique of excision and primary closure has a definite place in the management of this controversial disease.

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## Pseudomyxoma Peritonei†

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Pseudomyxoma peritonei is an interesting disease entity which has two principal sources, the ovary and the appendix. Statistically, ovarian cysts, benign and malignant, produce more pseudomyxoma peritonei cases than do mucocoeles of the appendix. The latter is a relatively rare cause of pseudomyxoma peritonei. According to Fritz, et al,<sup>1</sup> mucocoele of the appendix was first recognized by Rokitansky in 1842, Werth coined the phrase pseudomyxoma peritonei in 1884, and Fraenkel in 1901 first reported pseudomyxoma secondary to rupture of a mucocoele of the appendix.

The more common source for pseudomyxoma peritonei is ovarian tumors. From the Mayo Clinic, Cariker and Dockerty<sup>2</sup> reported 44 cases of pseudomyxoma peritonei arising from 138 malignant ovarian tumors. These authors made the interesting comment that the spill of the malignant cyst fluid at the time of surgery did not affect the prognosis adversely, whereas spontaneous rupture of the cysts for an undetermined length of time before surgery was serious. Thirty-five patients in their series died after spontaneous rupture of cysts. Shanks<sup>3</sup> reported 13 cases of pseudomyxoma peritonei in a series of 337 patients with benign and malignant ovarian pseudomucinous cysts.

### PATHOLOGY OF PSEUDOMYXOMA PERITONEI

The incidence of mucocoeles of the appendix has been reported as occurring from 0.07% to 0.2%. The asso-

ciation of peritoneal pseudomyxoma with or secondary to mucocoele of the appendix is extremely rare, only 2 cases having been seen in one hospital for 18 years during which time 13,000 appendectomies have been performed. Carleton<sup>1</sup> in 1955 reported the second case which had occurred at the Charity Hospital in New Orleans in 46 years. It is more commonly found in association with pseudomucinous cysts of the ovaries.

Pseudomyxoma peritonei is characterized by the finding of large amounts of mucoid material within the peritoneal cavity together with infiltration of the omentum and implants on the peritoneal surface by small cysts filled with mucin. The cysts may be lined with cells which vary from flattened endothelial cells to columnar epithelial cells or the cysts may have no epithelial lining.

Mucocoeles of the appendix arise from obstruction of the lumen. The mucosa is hyperplastic and papillary. The glands of Lieberkuhn are dilated and the hyperplastic epithelium overlying a stalk of connective tissue is compressed resulting in the papillary structures. In a late stage there may be atrophy of the epithelium.

The pathogenesis of pseudomyxoma peritonei is not clear but it is believed to result from perforation of the mucocoele. A fistula results which becomes lined with this hyperplastic epithelium and which then remains open allowing mucin to escape into the peritoneal cavity. The origin of the columnar cells lining the peritoneal cysts is in doubt; since they resemble the cells lining the mucocoele, one theory holds that they represent transplanted cells which then continue to secrete mucus. This theory has been disproved by

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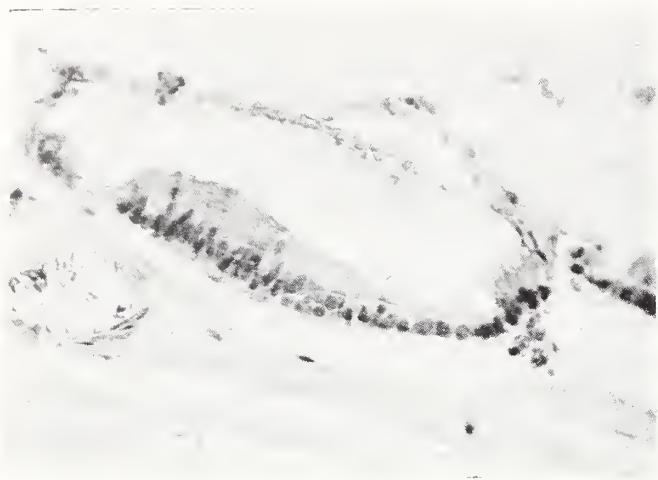


FIG. 1

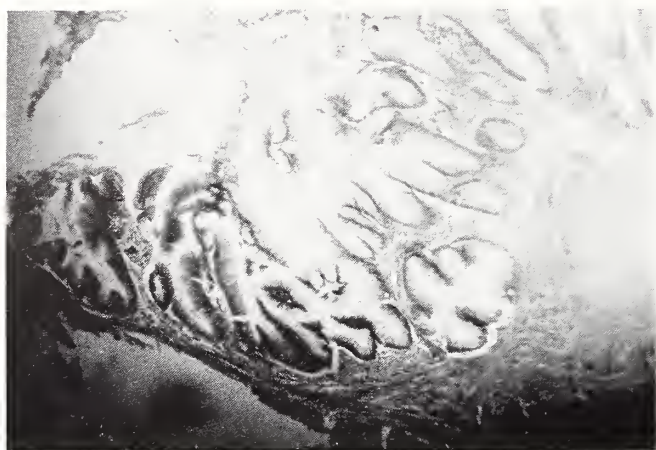


FIG. 2

Cheng's experiment in which appendiceal implants from an experimentally produced mucocoele in a rabbit failed to produce mucin and became walled off by fibrous tissue.<sup>1</sup> Transition of the mesothelial cells of the peritoneum to columnar cells has been observed as a reaction of irritation. The features of this disease are, therefore, the results of attempts of the peritoneum to localize the jelly secreted by the mucocoele. This theory received support from the fact that pseudomyxoma peritonei secondary to perforated mucocoele is improved or remains static following appendectomy.

In our case, the appendix shows the typical or characteristic findings of a mucocoele in that there is hyperplastic epithelium with papillary formation. The epithelium rests upon a muscle layer. The material resected at surgery and that found at postmortem examination within the abdominal cavity shows cystic spaces in which there is a partial lining of mucus secreting columnar epithelium. Separating these cysts are areas of dense fibrous tissue containing a scattering of lymphocytes. This epithelial lining shows no characteristics of malignancy.

The reason for progression of the disease process in this patient after removal of the appendix is presumed due to the advanced stage when first operated. It was impossible to remove all of the mucus and cysts. Those left behind continued to secrete mucus. The cause of death in these patients has been attributed to the disease

or to complications arising from surgery. This appears to be true in our case.

#### CASE REPORT

W. W. R. — This 68-year-old white man entered the hospital December 21, 1962, complaining of vomiting and stomach distress. He had had intermittent epigastric pain for at least a year which was usually relieved by food and soda. His pain had become steady in recent months and in the past month he had passed black stools and had had daily vomiting. His appetite remained good but he began to lose weight.

The patient had been in this hospital in February of 1959 and in 1961 because of cerebral thrombosis. He recovered satisfactorily. He was known to have had latent lues adequately treated in the past. On physical examination the patient appeared emaciated and quite ill, pale, and with a markedly protuberant abdomen bulging at the flanks. His diaphragm was high and did not descent on inspiration. The blood pressure was 120/82; the heart and lungs were normal. No peristalsis was audible. No abdominal masses were felt. His hematocrit was 36%, serum alkaline phosphatase was 7 Bodansky units. Blood electrolyte studies showed acidosis which was brought to normal in a few days by intravenous therapy. The BUN on 12/27/62 was 45 mg. %. Bilirubin was negative. The electrocardiogram was normal.

The patient was treated initially with intravenous fluids and gastric suction by way of a Levin tube. This brought him considerable relief from the symptoms but the symptoms recurred as soon as the suction was discontinued. The tentative diagnosis was probable carcinomatosis secondary to cancer of the stomach. Surgical exploration seemed indicated.

The patient was transferred to the Surgical Service and on December 28th exploration was carried out. The abdomen contained an estimated gallon or more of gelatinous fluid, brown and amber in color. An irregular (8.0 cm) mass of indurated tissue composed partly of gelatinous fluid was located in the greater omentum between the stomach and the colon. The visceral and parietal peritoneum was thickened and opaque in all areas making identification of organs difficult. No mass was palpable in the region of the appendix, in fact the whole right colon was invisible. Approximately one gallon of the gelatinous fluid was removed manually because the fluid clogged the suction apparatus. Also removed was the 8.0 cm. mass in the greater omentum. It was estimated that 75% of the gelatinous fluid was removed from the abdomen. The gross specimen consisted of a fibrous mass measuring 10 x 6 x 5 cm. and containing many small cysts filled with gelatinous material. Microscopically these cysts were lined with columnar epithelium. There was no pleomorphism of their nuclei and there was no cellular atypia. There were focal collections of lymphocytes within the fibrous stroma. The diagnosis was pseudomyxoma peritonei. (Fig. 1)

Convalescence from this procedure was amazingly satisfactory. He recovered his intestinal tone and was up and about the ward in good spirits. The advisability of reoperating on the patient and removing the appendix was determined. On February 25, 1963, at this secondary operation the appendix was found to be a mass 1.5 cm. in size, firm, white, and with the appearance of scar tissue or possible tumor. This appendix and/or tumor was removed. Three hundred to 400 cc. of gelatinous fluid were present in the abdomen at this operation. All of this fluid was removed insofar as possible. The appendix consisted of an irregularly shaped mass measuring 2 cm. in the greatest diameter. On one surface the stump could be recognized. On sectioning there was a central cavity 5 mm. in diameter containing mucin. Microscopically the irregularly shaped central cavity was lined with mucus secreting columnar epithelium which rested upon the muscle layer. There were papillary projections of the mucosa. Lymphoid tissue was absent. No perforation was seen. The changes were consistent with those of mucocoele of the appendix. (Fig. 2)



Again the convalescence was uneventful. The wound healed per primam. The patient was discharged March 25, 1963, for recall in 3 or 4 months.

The patient was readmitted on September 23, 1963, because of abdominal distention in the prior two months and vomiting in the prior week. The patient appeared well and alert. The abdomen was moderately distended. Fluid wave was demonstrated in the abdomen. No abdominal tenderness was present. The remainder of the physical examination was not remarkable.

The white blood count was 14,300 with 55% neutrophils. Stomach contents were 4-plus for occult blood. X-ray studies suggested four gall stones. On September 30, 1963, exploration was performed and several quarts of gelatinous fluid were removed and also a 6-8 cm. mass in the transverse mesocolon near the hepatic flexure. The wound was closed. Thirty mg. of nitrogen mustard were injected into the peritoneal cavity by way of catheters inserted into the peritoneum during the closing of the abdomen. The catheters were then withdrawn. The patient developed phlebitis of the left calf and a fecal fistula appeared on the twelfth postoperative day. The abdomen remained soft and the patient became mentally confused. The wound did not become generally infected. On the eighteenth postoperative day the temperature began to climb and on the twentieth postoperative day it reached 103.6° and the patient expired.

Postmortem examination was performed 6½ hours after death. The significant findings were limited to the abdomen. A fistulous tract extended from the right rectus operative scar to the ascending colon just above the cecum. The peritoneal cavity was largely obliterated by dense adhesions. There were several collections of nodular masses containing cysts filled with gelatinous material located about the spleen, over the right lobe of the liver, and in the pelvis. A culture of fluid from the right side of the abdomen grew out *Pseudomonas*. The stomach contained dark fluid which was guaiac positive. There were no mucosal lesions of the stomach or intestines. Microscopic examination of the nodular masses within the peritoneal cavity were characteristic of pseudomyxoma peritonei. There were no other findings of significance.

### DISCUSSION

The diagnosis of pseudomyxoma peritonei is not frequently entertained unless ovarian pseudomucinous cysts have previously been removed. Abdominal distention, intestinal obstruction, and the presence of ascitic fluid are all associated with pseudomyxoma peritonei and are non-specific signs. A correct preoperative diagnosis has been made by examination of pseudomucinous fluid removed by needle tapping of the abdomen.

Treatment of pseudomyxoma peritonei is surgical. If mucocoele of the appendix or ovarian cysts are removed before a rupture or spread, the prognosis may be good. For patients with ovarian origin of the disease, bilateral ovariectomy and hysterectomy are recommended by Shanks.<sup>5</sup> Appendiceal origin is treated by appendectomy. All the gelatinous implants are removed if possible. If

the disease has become extensive in the abdomen, repeated removals of fluid are indicated. X-ray therapy has not been recommended as being of benefit. Intraperitoneal nitrogen mustard was ineffective in our patient with pseudomyxoma peritonei.

The course of the disease is only too frequently progressive to a fatal ending. Repeated operative procedures over a period of several years may prolong the patient's life but recurring intestinal obstruction and resulting inanition and cachexia are the rule. Removals of large quantities of gelatinous fluid (4-10 liters) are reported. Shanks reports in 1963 four patients alive and well up to six years after surgery for ovarian cysts and pseudomyxoma peritonei. The patient with pseudomyxoma peritonei secondary to rupture of the appendiceal mucocoele is alive and well free of recurrence 3-1/2 years following surgery. Friedlaender<sup>3</sup> has put on record a four-year case without symptoms following appendectomy for giant mucocoele with two small cysts.

Pseudomyxoma peritonei secondary to appendiceal origin seems to have a slightly better prognosis than that of the ovarian precursors. Case reports are few, however. In our case, the appendix showed the typical or characteristic findings of a mucocoele. No perforation was observed but the appendix may have been perforated and subsequently encased in or obliterated by inflammatory reaction. A lack of improvement following appendectomy may then be explained by the large amounts of material within the peritoneal cavity which could not be completely removed and which continued to act as a source of irritation.

### SUMMARY

A case of pseudomyxoma peritonei due to mucocoele of the appendix is presented.

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# A Comparative Evaluation of an Alcoholic and A Non-Alcoholic Psychiatric Population\*

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The abuse of alcohol, or at least the recognition of this, has increased markedly in the past few years. The proliferation of local, state, national and even international, alcohol research and rehabilitation centers attests to the growing concern with this problem. As of 1962 the Classified Abstract Archive of the Alcohol Literature reported over 8000 articles dealing with this topic.<sup>3</sup> Keller<sup>6</sup> estimates that between four and five million Americans can be considered to be alcoholic. Adding to this figure another ten to fifteen million dependents of alcoholics, the number of people adversely affected by alcohol abuse mushrooms to between four-teen and twenty million.

Paralleling the marked increase in concern with alcoholism have been the speculations as to its etiology. The etiological hypotheses advanced center mainly in the areas of psychological, socio-cultural, physiological, and endocrinological factors—or admixtures of these.<sup>4,7,8</sup> The authors of this paper view the alcoholic as an individual who utilizes alcohol as a chemical reducer of the painful affects that attend his chronic inability to solve life problems. (Lienert and Traxel,<sup>9</sup> in a study evaluating the relative ability of meprobamate and alcohol to reduce emotionality, found both drugs to be equally better than placebo—the greater the emotional lability of the subject, the more effective the drugs. Anecdotal references to the tranquilizing properties of alcohol abound in the literature.) Although it appears likely that socio-cultural factors influence the choice of alcohol as a mode of dealing with stress<sup>1,12</sup> and that abuse of this agent may set off reaction patterns influenced by the physiology of the individual (which reaction patterns may precipitate a secondary cycle of problems) we hypothesize that the initial abuse of alcohol stems from the inability of the individual to cope constructively with life.

As part of a general research program at the Togus Neuropsychiatric Hospital Research Laboratory, all psychiatric patients admitted to this hospital are rated on certain demographic, historical, social and psychiatric variables. The scores are then coded on a Royal McBee coding card which provides us with a central source of data for all psychiatric patients. One of the variables on which data is collected is "presence of alcohol." A patient is scored positively on this item either when he admits to having a problem with alcohol or when, in the opinion of the examiner, the patient drinks to a

point where it interferes with his ability to maintain himself socially and/or economically in the community. Although others<sup>5,14</sup> have proposed quite complex classification systems for alcoholism, for the purposes of this paper an alcoholic is considered to be someone meeting the above criteria. While it may be argued that our alcoholic population is a selected one, i.e., they come to a psychiatric hospital, the findings of Plumeau et al<sup>13</sup> and Meer and Amon<sup>11</sup> that alcoholics in different settings cannot be differentiated along certain psychological dimensions, lends some support to extending these findings to the wider universe of alcoholics.

Between May, 1963 and November, 1963, 255 patients were admitted to our psychiatric hospital. Of these, 112 met our criteria of alcoholism (43%). The remaining patients (143) were grouped together to provide a comparative population. This latter population is a diagnostically heterogeneous psychiatric population. This paper presents an analysis of the distribution of the rated variables in both populations, comparisons of them, and some of the implications of these findings for the conceptualization and treatment of alcoholism.

Table I presents the demographic, social and historical data for the two populations. Column (1) describes the variables analyzed; columns (2) and (3) indicate the means or percentages for each variable in the alcoholic and non-alcoholic populations respectively; column (4) presents the inter-population comparison test utilized and obtained significance levels for these comparisons. Comparisons not significant at at least the .05 level are labeled NS (not significant).

It can be noted that the populations show no significant differences in the background variables of age, religion, number of children, education and length of military service. Of greater interest, however, is the similarity in the variables of percent from broken homes, occupation, work instability, and average number of NP admissions. The percent from broken homes in each group appears to be quite high, although no comparative figures for a non-psychiatric population were available. Comparing the occupational distribution of both groups with the occupational distribution of the non-hospitalized veteran population of Maine, we find that the latter population has 20.6% of their individuals in the professional-managerial group (the comparative figures for our hospital populations are 7.1% and 11.9% for the alcoholic and non-alcoholic populations respectively) and only 8.1% of their individuals in the unskilled group (compared to the study population figures of 25% and 23.1%). Both of our study populations

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TABLE I

DEMOGRAPHIC, SOCIAL AND HISTORICAL VARIABLES			
<i>Variables</i>	<i>Alco- holics</i>	<i>Non Alco- holics</i>	<i>Sig. Test</i>
Average Age	43	43	NS (T)
Protestant	53%	59%	NS (CHI.SQ)
Religion Catholic	45%	38%	
Other	2%	3%	
Percent from broken homes	28%	27%	NS
Percent never married	10%	21%	.05 (Z)
Percent divorced	40%	20%	.001 (Z)
Average number of children	2.25	2.19	NS
Average Education (years)	10.7	9.7	NS (T)
Average age of separation from parental home	22.4	25.7	.001 (T)
Average length of service (months)	40.9	37.0	NS (T)
Percent in Combat	30.4%	42%	.05 (T)
Occupation			
1. Professional-Managerial	7.1%	11.9%	NS
2. Clerical-Sales	12.5%	11.2%	NS
3. Service occupations	6.2%	5.6%	NS
4. Agricultural-Fishing	5.4%	6.3%	NS
5. Skilled	17.8%	12.6%	NS
6. Semi-skilled	21.4%	21.7%	NS
7. Unskilled	25.0%	23.1%	NS
8. Other	4.5%	7.7%	NS
Work Instability	47.3%	41.3%	NS
Average age at 1st NP admission	43.0	34.9	.001 (T)
Average total No. of NP admissions	3.20	3.01	NS
Average total length of NP stay (MONTHS)	8.1	13.8	.05 (T)
Average length of single hospitalization (MONTHS)	3.69	6.87	NS
Average number of GM&S hospitalizations	2.75	1.90	NS

are low on the socio-economic scale when compared with the non-hospitalized veteran population. The figures on work instability are even more startling. Using a similar definition of work instability as that employed with our study population, we found that 16% of the non-hospitalized veteran population of Maine had an unstable work history compared to 47.3% and 41.3% in our alcoholic and non-alcoholic populations, respectively. Both study groups show an equally high number of NP admissions.

The cogent variables which appear to differentiate the study populations are percent never married, percent divorced, age at first psychiatric hospital admission, and age at separation from parental home. The alcoholic group seems to be more likely to get married than the non-alcoholic psychiatric population (the comparative figure for the non-hospitalized veteran population of Maine is 12%). However, the individuals in the alcoholic group who marry have a much higher probability of getting divorced (40%) than either the individuals in the non-alcoholic psychiatric group (20%) or the non-hospitalized veteran population (4%). The non-alcoholic psychiatric population comes to a psychiatric hospital at an earlier age than does the alcoholic population. Also to be noted is that the alcoholic population

TABLE II

PSYCHIATRIC VARIABLES			
<i>Variables</i>	<i>Alcoholic %</i>	<i>Non Alcoholic %</i>	<i>Sig. Level</i>
1. Anxiety	72.3	56.0	.002
2. Guilt Feelings	58.9	42.0	.003
3. Conceptual Disorganization	8.9	22.4	.003
4. Unusual Thought Content	7.2	23.1	.0002
5. Mannerism and Posturing	4.5	11.9	.036
6. Emotional Withdrawal	22.3	31.5	NS
7. Blunted Affect	18.8	32.9	.014
8. Somatic Concern	44.7	39.9	NS
9. Tension	66.1	61.5	NS
10. Grandiosity	17.9	23.8	NS
11. Depressive Mood	70.5	67.8	NS
12. Hostility	19.6	16.1	NS
13. Suspiciousness	21.4	24.5	NS
14. Hallucinatory Behavior	19.6	21.7	NS
15. Motor Retardation	16.1	16.8	NS
16. Uncooperativeness	5.4	8.4	NS
17. Psychosomatic Involvement	61.0	54.0	NS

separates from their parental home at a significantly earlier age than does the non-alcoholic population.

The general impression gained from the comparisons of these variables is that our study populations are in many ways quite similar. Both groups present severe problems for the society in terms of their poor occupational preparation, their high divorce rate, unstable work histories, and their frequent hospitalizations. Although one can point out that the lower proportion of unmarrieds, higher average age at first psychiatric hospital admission, and lower age at separation from parental home in the alcoholic group makes this group appear to have a higher level of ego-integration than the non-alcoholic group, the former group's higher divorce rate and as frequent admissions to psychiatric hospitals, over time, attenuates somewhat the significance of this difference. From the vantage point of society, both of our study populations present serious social problems although the alcoholic psychiatric population as a whole presents these problems at a somewhat later age than the non-alcoholic psychiatric population.

Table II presents the data on the psychiatric variables rated in this study. Column (1) describes the symptoms rated; columns (2) and (3) indicate the percentage of patients in each group rated as showing these symptoms; and column (4) presents the significance levels for the inter-population comparisons. (The Z test was used for all comparisons here.) Both groups scored relatively high on those variables connoting uncompensated painful affects or their physiological equivalents, i.e., anxiety, tension, guilt feelings, depressive mood, somatic concern and psychosomatic involvement. However, the alcoholic population scored somewhat higher in these areas than did the non-alcoholic population, whereas the latter group scored higher on those variables more descriptive of psychotic symptomatology, i.e., conceptual disorganization, blunted affect and unusual thought content. The over-all impression obtain-

ed from these comparisons is that although both groups show a high incidence of psychiatric symptomatology, the symptom picture of the alcoholic population seems to be slightly less pathological than that of the non-alcoholic population. Generally speaking, the alcoholic group's psychiatric symptom pattern seems to resemble that of a severe neurotic population whereas the symptom pattern of the non-alcoholic population seems to fall at a point intermediate between that of a severe neurotic and psychotic population. (Connor,<sup>2</sup> comparing alcoholics to neurotics on a self-acceptance index, found no significant differences. MacAndrew, et al,<sup>10</sup> comparing male alcoholic outpatients with male non-alcoholic outpatients on certain scales of the MMPI, found no significant differences between these groups when items dealing specifically with alcohol use were dropped.)

The data obtained in this evaluation lends some inferential support to the position that our alcoholic population is in many respects similar to non-alcoholic psychiatrically disturbed populations. We view the alcoholic as attempting to mitigate the painful affects attending his inability to cope with life through the use of alcohol (a defense which over time seems to become less and less effective) the choice of this mode of stress reduction probably being less related to the nature of the problem than to other variables, e.g., socio-cultural patterns, physiological differences, etc. The social and economic dislocations provided by this mode of defense over time appear to be almost equal in scope to those produced by the defenses of the non-alcoholic psychiatric population even though the psychiatric symptom pictures of the two groups may differ somewhat along the dimensions described above.

Consistent with the position outlined above is the belief that conceptualizations about the alcoholic population should be similar to those employed with any psychiatrically disturbed population. Specifically, we support the view that the alcoholic differs importantly from other psychiatric patients only in respect to the defenses he employs against the painful affects produced by his chronic inability to deal with the basic psychological problems of life, and that his treatment should be similar to that of any psychiatrically disturbed population. From this viewpoint, unless we are specifically interested in symptom choice or the secondary problems

resulting from symptom choice, it does not seem parsimonious to set up research or treatment programs specifically geared for alcoholic populations. Rather, we see the alcoholic as but one more subtype under the generic heading of psychological disturbances who can be best understood and treated through an analysis of those factors that prevent any individual from dealing constructively with the demands of life.

#### SUMMARY

This paper presented a comparative evaluation of an alcoholic and a non-alcoholic psychiatric population on certain demographic, social, historical, and psychiatric variables. Some of the implications of these findings for the conceptualization and treatment of alcoholism were discussed.

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# Alcoholic Rehabilitation at the Veterans Administration Center, Togus, Maine\*

EDWARD V. MALCOM, PH.D. and PAUL F. FLETCHER, M.D.

## INTRODUCTION

For the purpose of focusing attention on the treatment of patients with alcoholic problems, a program of investigation and research was begun in the Psychiatric Service on January 5, 1963. Since the beginning of 1963, some 38% of admissions at this hospital were in some way significantly related to alcohol, and since then the excessive use of alcohol as a definite factor in admission histories has increased in ratio to non-alcoholic admission problems.

## METHODS AND PRELIMINARY RESULTS

The current program involved initially the setting apart of a unit of twelve selected and volunteer patients drawn from the hospital population on the basis of long-term addiction to alcohol. All had a history of some vocational success and this was considered a criterion for potential rehabilitation. At present the unit has a capacity for twenty patients at any one time (not necessarily the same individuals).

The program is broadly divided into two stages: In-hospital treatment and out-of-hospital, or follow-through. In-hospital treatment operates on a multi-therapeutic approach including (1) intensive group psychotherapy, (2) individual counselling or psychotherapy as needed, (3) hospital work assignments which are delegated according to the patients' capabilities and oriented towards building confidence and a sense of responsibility; all the facilities of the Physical Medicine and Rehabilitation Service are offered and patients are encouraged to fill their time with whatever meaningful tasks are available in the hospital organization; (4) vocational counseling is offered on an individual basis whenever required, (5) organized routine physical exercise and corrective therapy programs designed for physical conditioning and according to special needs of each patient, (6) medication as needed as an assist in stimulating and encouraging patients' participation in the above activities, especially when patients are depressed.

The out-of-hospital or follow-through part of the program includes informal communication by mail or telephone and by an "open door" policy for any patient who wants to talk over current tensions and problems. He is encouraged to feel welcome at any time to see a member of the staff, or even to join in one of the on-going groups during the daily therapy hour. A

timely visit can play an important part in warding off an alcoholic episode. We plan to make periodic visits to patients at their homes. This will be done either by a VA staff member or by a responsible community agency such as a representative of the State Alcoholic Rehabilitation Program, interested clergymen, or AA leaders. This activity is not yet fully operative.

Included in the out-of-hospital stage of the program is the treatment of the family of the patient to prepare more hopeful grounds for his return to family life. Other alcoholic programs have found that alcoholic patients with families have a generally better prognosis than the "loner," but the family must be included in the whole treatment process to increase the chances of success. At this time we encourage visits from wives as well as mothers or any other significant figures in the patients' lives to the hospital to discuss the family problem. The member of the family, say the wife, is seen by the staff member and usually with the patient.

The spouse is not always the "innocent victim" of the alcoholic husband's irresponsible and neglectful behavior. It appears that the wife may have entered the marriage to meet certain personal needs and the marriage may break up at the point where the alcoholic improves. Some wives become disturbed at the change of behavior in their husbands and may go all-out in an attack on his sobriety in the same manner as she did on his drinking. The wife sometimes must be helped to adjust to her non-drinking husband or find out why she resists the change.

Time limits for in-hospital and out-of-hospital stages of the treatment are set on an individual basis and in general will cover a total of from one to three years. During the out-of-hospital stage of the program, the patient is encouraged to seek out a counselor and even come back for a period of hospitalization without feeling that he has "failed." Since no "half-way house" is feasible for the foreseeable future in this area, the Alcohol Rehabilitation Unit must function in the position of both in-hospital and a "half-way house" facility.

We have now, in the Spring of 1964, entered a third phase of the program. Initially, in the first phase, considerable freedom was given the patient. A permanent privilege pass, easy access to town at any time, no retaliation or punishment for drinking (except when a nuisance to others), immediate placement on trial visit or discharge status on request of the patient were all features of a deliberate "laissez-faire" attitude in an attempt to avoid the role of "policeman" or finger wag-

\*From the Veterans Administration Center, Togus, Maine.

ging at the patient's alcoholic behavior. The idea was simply an attempt to minimize the effect of drinking as a device to get attention (albeit negative attention) and support. The freedom to come and go, to choose one's own hospital assignments, etc., was designed to offset the natural punitive reactions which the alcoholic arouses in other people. "Go ahead and drink, it's your life, your head, your choice." If a patient got too sick or annoying, he would be "dried out" and returned immediately to the unit. This phase of the program lasted eleven months and involved 36 patients passing through the 12-bed unit. Exposure to in-hospital treatment lasted from one day to the full eleven months. The average stay was three months. Over half of these patients have returned to their families or their work and have shown observable improvement in their alcoholic behavior pattern. About a quarter of the patients treated during this phase remain addicted and another quarter remain in the treatment program at present and appear improved.

Phase two of the program began in October 1963 (overlapping phase one) as a 20-bed ward became available for housing the patients. Phase two was characterized by a more restrictive and more directive treatment. The ward room could be locked as part of a locked ward or unlocked as part of an open ward. Privileges and passes had to be earned and could be easily lost. Assignments were arbitrary and often at the house-keeping level, even to designing "busy work" and non-creative, non-productive assignments, e.g. washing walls from one end to the other and then repeat the same for several days. Alcoholic behavior, upon release from these onerous assignments simply heaped on more dismal work for longer periods, or restrictions were placed, like a sentence, for a designated time limit.

Emerging from these two phases, and largely through an evolutionary process, we have entered a third phase of the alcoholic treatment program. Loss of privileges for the first "slip" was voted in by the patient group itself and the offending patient restricted from one to two weeks or until the group voted for the return of privileges. The second "slip" is considered a 30-day loss of privilege offense, and the third "slip" warrants 90 days before the group will consider a vote of return of privileges. Since January, only one patient has endured the 90-day restriction and he has visibly improved under it.

A significant part of the program from a research basis is an attempt to develop some predictor measures which would indicate for future development which patients respond best to what type of approach. It is understood that through our observations certain character disorder alcoholics do not respond satisfactorily to any but the most restrictive measures and perhaps are beyond the scope of this Veterans facility. The screening out of these individuals and the development of other types of programs would be subsequent to this research.

## DISCUSSION

### Basic Concept Relative to the Project

The basic concept of this program is based on the assumption that in certain, if not most, patients with alcoholic behavior problems emotional factors dominate and induce the alcoholic behavior. In other words, excessive drinking is not the alcoholic's main problem. Fear, rage, anger, and a deep sense of inadequacy seem to be important emotional factors which can be grouped according to

- (1) *Need for vengeance* (Targets: Past and present family, mother, father, wife, etc.)
- (2) *Exhibitionism* (Fantasy acting-out, "I'll show you," "look at me and feel sorry for me," "I'll make you pay attention," etc.)
- (3) *Need for self-punishment* (To assuage guilt for the hateful vengeance.)
- (4) *Need to reduce tension* (Derived from confusion of behavior, guilt, need for recognition, need for punishment, etc.) Fear plays a significant part in tension generation as well as low threshold for tolerating stress and anxiety. Alcoholics suffer from loneliness, isolation, dependency. They have unreal aspirations, and are beset by feelings of hostility, guilt, and depression.

Intensive group psychotherapy remains the *sine qua non* for all three phases thus far. Group therapy, usually led by two staff members, is directed toward uncovering and bringing to full awareness the negative emotional conditions the personal targets (people), and the use of alcohol as a weapon for attacking the target, inflicting punishment on oneself as a consequence of its use as a weapon, and as a formidable barrier to close personal relationships. The alcoholic behavior is used as a smoke screen and a potent repellant and shield much as other evasive and escape behavior patterns. Sobriety is considered a necessary means to arrive at proper and meaningful insights. Sanctions are imposed not only on drinking behavior but its easy ally—talking about drinking. "Slips" are analyzed in detail and within the framework of the four dynamic groupings to help the patient achieve a consciousness about his repetitive behavior patterns. Along with this, the patient must learn the process of reconditioning, of effective substitution, of constant vigilance for his own signals warning him of the growing tensions from which, in the past, he has sought escape through the use of alcohol. The process of therapy in this program includes ego-therapy, insight therapy, supportive therapy, chemotherapy within an orientation of encouraging self-reliance and reestablishing self-esteem. The goal is to regenerate self-motivation through an understanding of the principal of "freedom through responsibility" and the release of tensions tied up in archaic defenses. The inability or unwillingness of some individuals to accept either responsibility or the need to detach themselves from archaic defense patterns necessitates the inclusion



of the restrictive approaches which may be needed in some cases not so much to rehabilitate the patient, but to withdraw him from circulation as a public nuisance and slow down the "yo-yo" effect of bouncing from the community to the admission ward. The designation of this type of patient (and his control) is one of the objectives of the research program.

#### MEASURES OF SUCCESS

"Success" in this rehabilitation program will be judged not so much in terms of "sobriety" maintained by the patient but rather by change of attitudes and the more efficient and creative consequences of his expanded energy. It would be assumed that sobriety will follow the more positive approach to living but not necessarily on abstinence from drinking based on old patterns of negative feelings ("I stayed sober for four years out of sheer bull-headedness!" stated one of our more chronic patients.) A form of success would certainly be judged on the ability to seek less harmful substitutes than alcohol for him and seek personal help when he recognizes the signs of distress which at one time would have driven him to reach for the bottle.

The focus of research for this rehabilitation program includes (1) the selection of patients according to the category of emotional disorders inducing or underlying the alcoholism, (2) the follow-through or out-of-hospital stage of the program dealing with the immediate family environment of the patient and their treatment, and the "follow through" of the patient himself when he is ready to attempt living in the community.

"Success" of the program is being measured by comparing times and length of hospitalizations before and after exposure to the program (some patients are on

their first admission). It is also based on the patients' return to economic productivity. In the follow-through stage the purpose is to investigate the long-range therapeutic gains which can be made beyond the therapeutic gains the patient achieved in the hospital.

A word on Alcoholics Anonymous: This program in no way opposes or contra-indicates the AA program as it is widely known today. In AA many alcoholic behavior patterns are arrested successfully as the individual regains his lost self-esteem and breaks through his isolation. Many alcoholics continue to function adequately with the help of AA. Patients in our program are encouraged to look into AA as one source of support and as a means of maintaining sobriety especially after leaving the unit.

#### CONCLUSION

It must be emphasized that no quick and easy solution has been discovered for the problem of "alcoholism." Although Togus, in a sense, is "pioneering" in this area, its program is nevertheless based on the insights and contributions to the field of alcoholic studies emerging from such alcoholic centers as Massachusetts General Hospital, Chicago's Warren Clinic, The Cleveland Center for Alcoholism, and Yale and Rutgers Universities' researchers in alcoholism.

Sweeping away the dust of prejudice about alcoholism and adapting these programs as well as our own findings to the special needs of the Maine community is the task which the staff at Togus has set for itself through the current alcoholic program.

The conclusions reported in this paper are tentative only. Further attempts will be made to evaluate these methods of treatment and results will be reported.



# The Impact of an Aging Population on a Neuropsychiatric Veterans' Hospital

DAVID W. BRIGGS, PH.D.\*

It is well-known that the veteran population, based as it is primarily on the World War I and World War II veterans, is growing older. This change magnifies for the veterans' hospitals the problems created by increasing longevity with which the nation is now faced. A distribution by age of the inpatient NP hospital population at Togus is bimodal with the 35-44 and the 65-74 age groups as peaks. This reflects the WWI and WWII veteran populations. Approximately 30% of this population is 65 or older. In order to clarify the extent of change in patient characteristics which come with age, and the care they required due to changes, the following survey has been made.

It is hypothesized that with increasing age, NP patients will generally show a deterioration in condition and an increasingly poor prognosis. More specifically, it is postulated that with increasing age, (1) the nursing care for physical needs will increase; (2) the over-all level or adequacy of social functioning will decrease; and (3) the prognosis will become poorer for improvement, length of hospitalization, and the level of extra-hospital placement.

## PROCEDURE

Samples of 20 patients from each of six age groups (0-34, 35-44, 45-54, 55-64, 65-74, 75+) were randomly selected for rating. These patients were rated on five scales by the nurses on the wards.

## RESULTS

### 1. Nursing Care for Physical Needs

The frequency of patients requiring at least "minimal" nursing care increases steadily from 30% for those under 35, to 80% for those 75 and over. Since patients who are seriously ill or handicapped physically are likely to be treated and classified as GM&S patients, there

are only a few in any age group considered to require a moderate or high degree of care.

### 2. Adequacy of Social Functioning

The frequency of patients whose over-all level of social functioning is rated as "good" or "very good" decreases from 75% for those under 35 to 10% for those 75 and over.

### 3. Prognosis for One Year

Although nurses predict conservatively that most patients of all age groups will be the same a year hence, the prediction of "improved" drops from 40% for those under 35, to 0% for those 75 and over, and the prediction of "deteriorated" increases from 5% to 40%.

### 4. Predicted Length of Hospitalization

The prediction of "prolonged hospitalization" (over one year or terminal) increases sharply with age from 5% for the youngest group, to 75% for those 55-64, to 90% for those 75 and over.

### 5. Possible Extra-hospital Placement

The level of "extra-hospital placement" considered to be possible deteriorates. Fifty percent of those under age 35 are viewed as potentially self-supporting but beyond the age of 55 (well below the culturally accepted retirement age of 65) patients are viewed as seldom capable of self-support. Placement in a supervised home or foster home setting is seen to be a reasonable solution for about one-third of patients of all age groups. The frequency of patients rated "probably will be unable to leave the hospital" increases from 10% for the youngest group, to 50% for the oldest.

## DISCUSSION

The results support the hypotheses regarding the relationship of age to patients' condition and prognosis and demonstrate that age is an important predictive variable. With an aging patient population, it is evident that there will be an increasing need for facilities to care for, to treat, and to place in sheltered environments the chronic cases with limited potential for rehabilitation.

\*Counseling Psychologist, Veterans Administration Center, Togus, Maine.





DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### Implementation of Kerr-Mills Medical Aid\*

NILES L. PERKINS, M.D.\*\*

The Kerr-Mills Program (Medical Assistance for the Aged) is the compromise legislation which has been supported by the American Medical Association in the hope that it would stop pressure for socialization of medicine.

Passed by Congress in 1960 in place of the so-called King-Anderson Bill, the Kerr-Mills Bill was sponsored by two Congressmen (both Democrats) who did not want medical care tied to the Social Security program. Now why was this bill so important and why did our Medical Association endorse it?

1) This bill did not tie medical care in with the Federal Social Security Plan and payments made by the Federal Government come from the general budget. Furthermore, it is a comprehensive plan which allows states to set up almost any program they want or can afford to support with a share of state funds.

2) The Kerr-Mills Program is preferable, in my opinion, because it gives the states a free hand in setting up their own medical programs. This includes the right of the state to determine the terms of eligibility, the length of hospital stay, and the various other medical needs aside from hospitalization which can be included in accordance with the degree of contributory state funds.

However, because the cost of this plan, in part, comes out of each state's general budget, only 36 states have started the MAA program. The other states are waiting, perhaps because of lack of leadership in this direction, or for a Medicare bill tied to the Social Security Program which supposedly would not cost the states anything. This, however, has to be taken with a grain of salt if our Medicare Program ends up anywhere near as expensive as the British National Health Plan.

#### NOT ALL ROSY

The major problem with the MAA plan is that most

of the money spent by our government has gone to the six or eight richer states because they appropriated more money for their share, or simply used federal MAA money to replace state expenditures in existing programs; for example, nursing home care for old age assistance recipients. They also have much more comprehensive programs. Obviously, the matching funds from the Federal Government are much less than in the states with a lower per capita income. This was an attempt by Senators Kerr and Mills to induce the states with less money to initiate the plan on their own volition.

At the present time in Maine, 65-70% of the money spent on MAA is federal money which comes from Washington and does not come out of the state's budget.

Eligibility requirements vary from state to state almost as much as the scope of services. The states can set the limit of income and assets at almost any level in their determination of the medically indigent.

#### THESE PATIENTS ARE NOT PAUPERS

One of the more important points in our state plan is the absence of relative responsibility for a patient's hospital bill which some states do have. This helps preserve the dignity of the patient who is self-sufficient in his maintenance but can be quickly overcome with a period of illness.

#### WHAT DOES THE MAA PLAN COVER IN MAINE?

At present our program pays for only hospitalization for reasonable periods. It does not cover terminal care over an extended period, nor does it cover nursing home care at the present time. It does cover limited clinic services where such clinics are available.

There just isn't enough money under the Maine plan to pay for the many other kinds of medical services which many elderly persons need — such as drugs, physician's home visits, transfer to nursing home care — unless in such transfer the patient becomes eligible for Old Age Assistance. Under the latter, however, there are more stringent regulations on property, income and relative responsibility which are more likely to embarrass the patient.

\*This presentation is reprinted from a paper given by Dr. Perkins at a medical group meeting. It expresses his personal views and does not necessarily reflect any position taken by the Department of Health and Welfare.

\*\*State Consultant on Medical Assistance for the Aged.

### PAYMENTS TO HOSPITALS

Present payments to hospitals are scaled to the size and scope of hospital services and length of patient stay. The larger hospitals receive \$26.00 a day for all-inclusive services during the first ten days. Then \$20.00 a day for the next 35 days and finally dropping to \$15.00 for the rest of the patient's stay. The rest of the hospitals get \$20.00 a day for the first ten days, then \$15.00 a day for the next 35 days and finally down to \$12.50 a day for the remainder.

To meet federal requirements clinic visits are included. However, at the present time, this service is available only at the Thayer Hospital in Waterville and the Maine Medical Center in Portland.

Under the Kerr-Mills plan, federally, it is permissible to pay for medical services by a doctor, but it was the opinion of the Maine Medical Association's Advisory Committee on the Kerr-Mills Program that doctors should not accept fees of this nature until all efforts had been taken to get the Medical Assistance for the Aged plan working smoothly and there was a good comprehensive plan in effect. It was also felt that acceptance of a fee of this nature, which was not a negotiated fee, was kin to Socialized Medicine.

One point should be made clear about the Medical Assistance for the Aged Plan. It has nothing to do with the control of relationships between the doctor and patient. The patient can have free choice of his doctor and the doctor can charge the patient for services rendered as he normally does. The hospitals, however, because most are receiving from the state an all-inclusive rate which is below their ward cost, usually stipulate that the patient has to be hospitalized in a ward bed unless there are true medical indications, such as infectious disease or a severe medical problem.

### HOW FAR-REACHING

It is now estimated that in the State of Maine there are at least 25,000 persons eligible under the Medical Assistance for the Aged Plan. About 6,400 have already been certified. Certification is advised for all who may be eligible so as to avoid confusion, delays or worry in time of illness.

Anyone 65 years of age or over, who has an income

of less than \$1,500 per year if single, or \$2,100 if married and who may have, in addition to income, assets of equal value, may apply for certification. They may own their own home with no strings attached. Their assets may be in addition to the home in which they live. Naturally, they must be residents of the state for at least one year. These requirements in Maine are comparable to the eligibility levels, services, etc. in other states.

One mistake sometimes made by doctors is to consider the Medical Assistance for the Aged program now in effect as demeaning or pauperizing. It is not. It is far more liberal than Old Age Assistance and state and federal law prohibit pauperizing.

This aid may be applied for at time of hospitalization, but advance application is urged by simply writing to the Department of Health and Welfare, Augusta, or inquiring at the nearest district office, hospital or municipal office. Certification must be renewed annually.

### USAGE AND PHYSICIAN PARTICIPATION

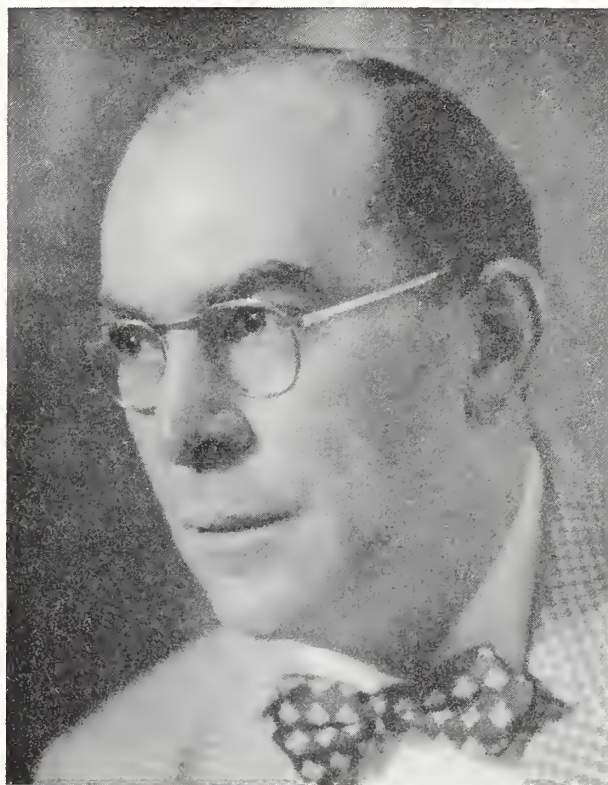
During 1963 there were between 250 and 300 patients in Maine hospitals each month under this plan. The state has budgeted \$1,500,000 for the current fiscal year, of which 34% will come from state funds and the balance from the federal government.

You have already heard about the statewide Medical Advisory Committee which was established a year ago and is going ahead steadily in a variety of important studies. This Committee, whose chairman is Dr. George E. Sullivan, Waterville, is already proving that doctors can sit down together and develop a medical care program in their own locale and then orderly police themselves. The Committee is still young and has a long way to go, but it's on the road and we are much encouraged by the interest and participation shown.

To conclude, may I point out that it is up to every doctor to help implement the Medical Aid for the Aged program made possible by the Kerr-Mills Act in an attempt to cut down on the further legislation of socialized medicine; and also to become involved as much as possible at the local level in the administration of this plan so that it does not in itself become a socialistic medical monster.



*President*  
*Maine Medical Association*  
1964 - 1965



THOMAS A. MARTIN, M.D.

THOMAS A. MARTIN, M.D. of Portland, Maine assumed his duties as President of the Maine Medical Association on June 15, 1964 at the 111th Annual Session banquet. Dr. Martin served as Councilor for the First District from 1960 to 1963, the last year as Council Chairman and as President-Elect from 1963 to 1964.

Dr. Martin was born in Biddeford, Maine on October 14, 1905, the son of John and Ellen Buckley Martin. He was educated at the elementary schools in Biddeford, Maine and was graduated from Biddeford High School in 1923, the University of Maine in 1929 and received his medical degree from Tufts University Medical School in 1932. He interned at the Maine General Hospital from 1932 to 1933; did postgraduate study in Orthopedics at the University of Pennsylvania Graduate School from 1936 to 1937 and served a residency in Orthopedics at the St. Luke's Hospital in Kansas City, Missouri from 1937 to 1938. Dr. Martin is Chief of Orthopedic Services at the Maine Medical Center and the Mercy

Hospital in Portland and is Consultant to the Portland City Hospital.

Dr. Martin is a member of the American Medical Association, the Maine Medical Association and the Cumberland County Medical Society. He is also a Diplomate of the American Board of Orthopedic Surgery, a Fellow of the American Academy of Orthopedic Surgeons, a Fellow of the American College of Surgeons and a member of the Bone and Joint Association.

He is a life member of the Tufts University Alumni Council and is serving a third four-year term on the Tufts University Medical Alumni Council of which he was elected Vice-President in March 1964. He is also a member of the Editorial Board of the Bulletin, the Tufts Medical School Alumni publication.

Mrs. Martin is the former Clovis A. Hawkes, R.N. of Portland, Maine, and they have two sons, Dr. Thomas A. Martin, Jr., a graduate of Tufts Medical School in 1964, and Peter I., and a daughter, Ellen C.

# Maine Medical Association

## STANDING COMMITTEES 1964-1965

Standing Committees for 1964-1965 as proposed by the Nominating Committee and approved at the Second Meeting of the House of Delegates of the Maine Medical Association at Rockland, Maine, June 14, 1964.

### Nominating Committee

- 1st District* – JOHN F. GIBBONS, M.D., Portland – *Chairman*  
*2nd District* – MORRIS E. GOLDMAN, M.D., Lewiston  
*3rd District* – HARRY G. TOUNGE, JR., M.D., Camden  
*4th District* – SAMSON FISHER, M.D., Waterville  
*5th District* – RUSSELL G. WILLIAMSON, M.D., Blue Hill  
*6th District* – JOHN B. MADIGAN, M.D., Houlton

### Scientific Committee

- Albert L. Hunter, M.D., Camden Community Hospital, Camden (1 yr.) – *Chairman*  
 Richard P. Laney, M.D., 50 Water St., Skowhegan (2 yrs.)  
 Clement A. Hiebert, M.D., 18 Bramhall St., Portland (3 yrs.)

### Legislative Committee

- John D. Denison, M.D., 105 Brunswick Ave., Gardiner (1 yr.) – *Chairman*  
 Howard P. Sawyer, Jr., M.D., 22 Bramhall St., Portland (2 yrs.)  
 Kevin Hill, M.D., 33 College Ave., Waterville (2 yrs.)  
 Warren G. Strout, M.D., 205 French St., Bangor (3 yrs.)  
 Michael J. Harkins, M.D., 437 Main St., Lewiston (3 yrs.)

### Committee on Recruitment, Aid and Placement

- Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville (2 yrs.) – *Chairman*  
 Merle S. Bacastow, M.D., 22 Bramhall St., Portland (2 yrs.)  
 Peter B. Aucoin, M.D., 151 Franklin St., Rumford (1 yr.)  
 George W. Wood, III, M.D., 156 No. Main St., Brewer (3 yrs.)  
 Charles A. Hannigan, M.D., 85 Goff St., Auburn (3 yrs.)

### Medical Advisory Committee

- Thomas A. Martin, M.D., 157 Pine St., Portland – *Chairman*  
 Gerald H. Donahue, M.D., 4 Station St., Presque Isle  
 Philip L. Gray, M.D., Blue Hill  
 C. Harold Jameson, M.D., Medical Arts Bldg., Rockland  
 George L. Maltby, M.D., 31 Bramhall St., Portland  
 Clement S. Dwyer, M.D., 205 French St., Bangor  
 Allan J. Stinchfield, M.D., P.O. Box 343, Augusta

### Public Relations Committee

- Robert J. Barrett, Jr., M.D., Cor. Union & James Sts., Bangor (2 yrs.) – *Chairman*  
 Lane Giddings, M.D., 6 E. Chestnut St., Augusta (1 yr.)  
 John A. Root, M.D., 22 White St., Rockland (1 yr.)  
 John R. Lincoln, M.D., 22 Bramhall St., Portland (3 yrs.)  
 John B. Madigan, M.D., Houlton (3 yrs.)

### Board of Ethics and Discipline

- Harry Brinkman, M.D., 47 Perham St., Farmington (2 yrs.) – *Chairman*  
 John E. Whitworth, M.D., 336 Mt. Hope Ave., Bangor (2 yrs.)  
 William F. Mahaney, M.D., 338 Main St., Saco (1 yr.)

- Ralph A. Goodwin, Sr., M.D., 56 Denison St., Auburn (1 yr.)  
 Emerson H. Drake, M.D., 18 Bramhall St., Portland (3 yrs.)  
 Robert L. Ohler, M.D., Veterans Administration, Togus (3 yrs.)

### Rural Health Committee

- Paul A. Fichtner, M.D., 781 High St., Bath (2 yrs.) – *Chairman*  
 A. Dewey Richards, M.D., 11 Gage St., Bridgton (2 yrs.)  
 Donald E. Allen, M.D., Sebago Lake (1 yr.)  
 Alexander W. Magocsi, M.D., York (1 yr.)  
 Mary M. Dietrich, M.D., P. O. Box 93, Orrington (3 yrs.)  
 John Kazutow, M.D., Box 113, Columbia Falls (3 yrs.)

### Committee on Credentials

- Paul E. Floyd, M.D., 2 Middle St., Farmington (2 yrs.) – *Chairman*  
 Merrill J. King, Sr., M.D., 129 Pine St., Portland (2 yrs.)  
 Thomas G. Harvey, M.D., 59 Mayo St., Caribou (1 yr.)  
 Russell M. Lane, M.D., Water St., Blue Hill (3 yrs.)  
 Nelson P. Blackburn, M.D., Bath Memorial Hospital, Bath (3 yrs.)

### Health Insurance Committee

- Francis A. Winchenbach, M.D., 910 Washington St., Bath (3 yrs.) – *Chairman* (Lincoln-Sagadahoc)  
 George B. O'Connell, M.D., 11 Lisbon St., Lewiston (2 yrs.) – (Androscoggin)  
 George J. Harrison, M.D., Market Sq., Houlton (2 yrs.) – (Aroostook)  
 Albert Aranson, M.D., 39 Deering St., Portland (3 yrs.) – (Cumberland)  
 Gunther H. Rowe, M.D., 42 Main St., Livermore Falls (1 yr.) – (Franklin)  
 Robert F. Russell, M.D., Penobscot (2 yrs.) – (Hancock)  
 Kenneth W. Sewall, M.D., 2 School St., Waterville (2 yrs.) – (Kennebec)  
 Edward K. Morse, M.D., 22 White St., Rockland (2 yrs.) – (Knox)  
 Ake Akerberg, M.D., 2 E. Main St., South Paris (1 yr.) – (Oxford)  
 Leonard G. Miragliuolo, M.D., 10 Maple St., Bangor (1 yr.) – (Penobscot)  
 Charles H. Lightbody, M.D., No. Main St., Guilford (3 yrs.) – (Piscataquis)

*Continued on Page 142*



## *Maine Heart Association Notes*—————



### **Reevaluation of Therapy of Acute Myocardial Infarction**

“The rate of mortality from acute myocardial infarction has not declined commensurately with the general advancement of medical technology. Anticoagulant therapy, which has decreased the incidence of thromboembolic complications only slightly and the mortality rate even less, has perhaps distracted the clinician from the search for more successful therapeutic approaches. . . .

“Intensive-care units which utilize modern electronic monitoring, therapeutic devices, and medical personnel trained and drilled in the procedures for cardiac resuscitation are considered to be an urgent need. Intensive-care units are likely to be the best means now available to clinicians for decreasing the rate of mortality from acute myocardial infarction.”

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Reference: Lindsay, M. I., Jr. and Spiekerman, R. E. *American Heart Journal*, Volume 67, pages 559-564, 1964.

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### **American Heart Association's 37th Scientific Sessions and Annual Meeting at Atlantic City**

Registration forms may now be obtained for the Scientific Sessions to be held at the Atlantic City Convention Hall, October 23-25. Six half day clinical sessions have been designed to meet the needs of practising physician and simultaneous sessions are scheduled for presentation of papers based on original investigative work in the cardiovascular field. The Annual Meeting will be held October 25-27 at the Hotel Chalfonte — Haddon Hall, Atlantic City.

For complete program and registration forms, contact the Maine Heart Association, Augusta, Maine or the American Heart Association, 44 East 23rd Street, New York City, N. Y. 10010.

## County Society Notes

### PENOBSCOT

A meeting of the Penobscot County Medical Society was held at The Oronoka Restaurant in Orono, Maine on April 21, 1964. The President-Elect, Richard T. Munce, M.D., presided.

The speaker of the evening was Robert E. Wise, M.D. from the Department of Diagnostic Radiology of the Lahey Clinic. He discussed Current Concepts of Cholangiography and Angiography at the Lahey Clinic.

Philip B. Thomas, M.D. reported on the Insurance Program now held by the Penobscot County Medical Society. A motion was made by Clement S. Dwyer, M.D. that the report of Dr. Thomas be accepted for the minutes of the current meeting for informational purposes and voted upon at the next regular meeting.

Arthur N. Lieberman, M.D. reported on the Interim Meeting of the Maine Medical Association House of Delegates. He discussed the budget, a medical school in Maine, present difficulties with the Medicare contract, the report of the Maine Medical Education Foundation and the need for election of a Councilor from the Sixth District by the society at this meeting.

Asa C. Adams, M.D., of Orono was chosen as the nominee for Councilor for the Sixth District (Aroostook, Piscataquis and Penobscot County Societies). (Note: A caucus of the delegates from these three counties resulted in Dr. Adams' name being presented at the annual meeting of the House of Delegates as Councilor for this District and he was elected for a three-year term).

The monthly meeting of the Penobscot County Medical Society was held on May 15, 1964 at the Penobscot Valley Country Club in Orono, Maine. The President, Richard T. Munce, M.D., presided.

Dr. David Karnowsky of Memorial Hospital and Sloan Kettering Institute, New York City presented an interesting paper on Cancer Chemotherapy.

HADLEY PARROT, M.D.  
*Secretary*

### SOMERSET

The regular meeting of the Somerset County Medical Society was held at the Village Candlelight in Skowhegan, Maine on April 21, 1964. The President, W. Edward Jordan, Jr., M.D. called the meeting to order.

Ernest W. Stein, M.D., President of the Maine Medical Association, spoke on the passing of Ernest D. Humphreys, M.D. of Pittsfield who died on March 15 of this year. The President appointed Dr. Stein to write a eulogy to Dr. Humphreys with copies being sent to the widow and the Maine Medical Association.

George E. Sullivan, M.D. reported on the April 12 Interim Meeting of the Maine Medical Association House of Delegates and among other matters brought to the attention of the society the newly proposed budget. A motion was made and seconded that the society pass the budget.

Edgar J. Smith, M.D. read a letter dealing with Medicare benefits and payments to physicians. In essence the letter suggested that fees should be reduced even below Blue Shield level. After some discussion, a motion was made that Dr. Smith contact Dr. Hanley about renegotiation of the contract and advise him that the members do not accept the decrease as proposed.

George J. Robertson, M.D., formerly of Waterville, and now at the New England Center Hospital was the guest speaker. His subject dealt with the newer antibiotics of the penicillin

group especially, such as prostaphlin and polycillin. Accompanying Dr. Robertson was Dr. Anthony Lewis who is Regional Advisor in Postgraduate Medicine at the University of London. He was in this country studying postgraduate education, being a Postgraduate Fellow of the Commonwealth Fund.

Following adjournment the members and wives enjoyed a social hour and dinner.

MARIAN L. STRICKLAND, M.D.  
*Secretary*

### KENNEBEC

The annual guest meeting of the Kennebec County Medical Association was held at the Augusta Country Club in Augusta, Maine on May 21, 1964. Sixty-six members and guests were present at the meeting which was called to order by the President, Kenneth W. Sewall, M.D. This was followed by a social hour and dinner.

Leonard W. Cronkhite, Jr., M.D., the guest speaker, spoke on the medical aspects of space travel. Dr. Cronkhite is general director of the Children's Hospital Medical Center in Boston and is also involved in research and development with several industries in the Boston area concerned with space travel. He spoke of the various deprivations the astronauts must undergo on their space flights and explained in part the method of choosing the astronauts.

EARLE M. DAVIS, M.D.  
*Secretary*

### CUMBERLAND

The regular monthly meeting of the Cumberland County Medical Society was held on May 21, 1964 at the Holiday Inn in Portland, Maine with 92 members and guests attending.

Following a social hour and dinner, the society watched part of a television program sponsored by the AMA entitled "The Making of a Doctor."

Boris A. Vanadzin, M.D. discussed his proposal for abolishing school immunization clinics in favor of centralized mass clinics, allowing more efficient use of professional personnel and making public awareness of the clinics more effective. After a general discussion, Charles R. Geer, M.D. stated that it was his opinion that the society was in support of Dr. Vanadzin's proposal.

Dr. Vanadzin then presented his final report on the Health Careers Week program, reading comments by participating physicians and students, all of whom seemed enthusiastic about the undertaking. He offered three specific recommendations:

1. That only high school seniors and juniors should participate in this field project.
2. That the project should again be scheduled in the spring vacation.
3. That other health groups should participate so as to allow the exposure of students to other than the medical practitioners.

There was discussion of the proposal made at the April meeting of the House of Delegates of the Maine Medical Association to amend the constitution so as to allow only one scientific session a year. The membership voted to instruct its delegates to support this resolution. The members then voted to encourage assessment rather than voluntary contribution of the \$25.00 sum for the Maine Medical Education Foundation in 1965, should the matter be brought up at the June meeting of the House of Delegates.

*Continued on Page 142*



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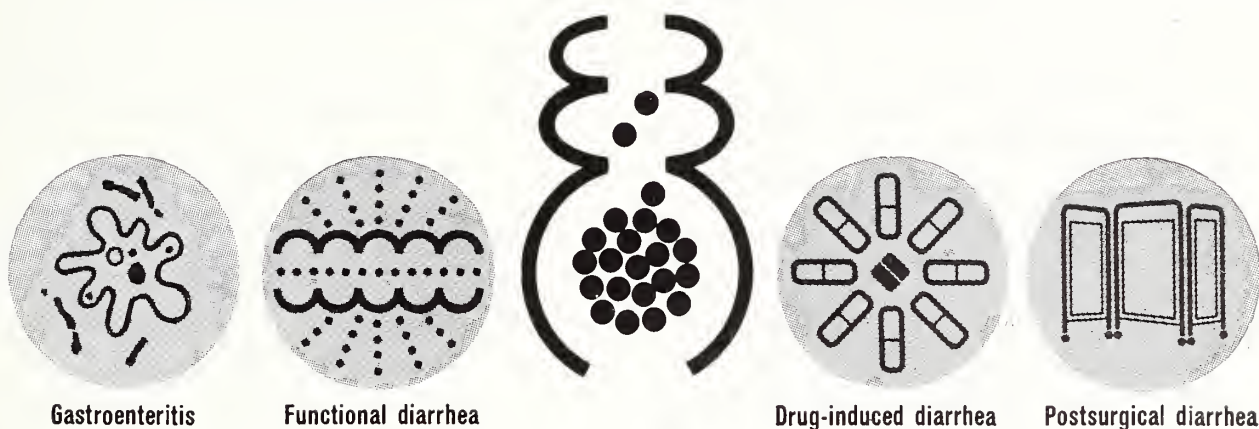
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**SEARLE**

COUNTY SOCIETY NOTES — *Continued from Page 140*

Dr. Geer mentioned Mr. Matthew Barron's desire to address the Cumberland County Medical Society regarding the Kerr-Mills Bill. The membership expressed interest in the matter but a lack of enthusiasm over a special summer meeting for this purpose.

STANLEY B. SYLVESTER, M.D.  
*Secretary*

## WASHINGTON

A regular meeting of the Washington County Medical Society was held in conjunction with the St. Croix Medical Society on June 9, 1964 at the Charlotte County Hospital in St. Stephen, New Brunswick. Sixteen members and guests were present.

The speaker for the evening was Dr. Lea C. Steeves, cardiologist from Halifax, Nova Scotia. Cases were presented by Dr. W. C. Rice of Milltown, New Brunswick; Dr. Donnie Smith of St. Andrews, New Brunswick and Dr. E. B. Johnston, of St. Stephen, New Brunswick. These cases were all discussed by Dr. Steeves, with the various aspects of diagnosis and treatment being discussed by the attending physicians.

The meeting was followed by a short business meeting of the St. Croix Medical Society and of the Washington County Medical Society. The Washington County Medical Society was presided over by the President, James C. Bates, M.D. The last interim meeting of the Maine Medical Association House of Delegates was discussed, particularly, the new Medicare contract. It was voted to hold a meeting in July of both societies for business only.

KARL V. LARSON, M.D.  
*Secretary*

## YORK

The bimonthly meeting of the York County Medical Society was held at the Notre Dame Hospital in Biddeford, Maine on

June 10, 1964. The meeting was called to order by the President, Roger J. P. Robert, M.D., following a social hour and dinner.

Alvin A. Hoffman, M.D. was appointed to look after the October meeting to be held at the York Hospital.

It was voted to instruct delegates to vote to do away with the Fall Clinical Session of the Maine Medical Association.

Mr. Elmont S. Tyndale of Kennebunkport was the guest speaker. He gave a very interesting and instructive talk on Legislation and the M.D. A very interesting question and answer period followed.

CHARLES W. KINGHORN, M.D.  
*Secretary*

## New Members

## ANDROSCOGGIN

Lionel R. Tardif, M.D., 128 Sabattus St., Lewiston

## CUMBERLAND

Martin A. Barron, Jr., M.D., 37 Deering St., Portland  
Winton Briggs, M.D., 507 Ocean St., South Portland

## KNOX

Harry A. Naumer, M.D., Land's End, Port Clyde

## PENOBSCOT

William M. Blackwell, M.D., Millinocket Com. Hosp., Millinocket

## WALDO

Ward A. Albro, M.D., 1 Church St., Belfast  
George M. Malouf, M.D., Islesboro

STANDING COMMITTEES 1964-1965 — *Continued from Page 138*

Edgar J. Smith, M.D., 1 Park St., Fairfield (1 yr.) — (Somerset)  
George L. Temple, M.D., Fahey St., Belfast (3 yrs.) — (Waldo)  
James C. Bates, M.D., Eastport (1 yr.) — (Washington)  
Roger J. P. Robert, M.D., 331 Main St., Saco (3 yrs.) — (York)

### Members of the Advisory Committee to the Health Insurance Committee

Maine Society of Anesthesiology — Clement S. Dwyer, M.D., 205 French St., Bangor  
Maine Chapter, American Academy of General Practice — John D. Denison, M.D., 105 Brunswick Ave., Gardiner  
Maine Society of Obstetrics and Gynecology — E. Allan McLean, M.D., 29 Deering St., Portland  
Maine Chapter, American Academy of Pediatrics — Everett A. Orbeton, M.D., 131 Chadwick St., Portland  
Maine Society of Internal Medicine (Includes Medical Specialty Group) — Albert Aranson, M.D., 39 Deering St., Portland  
Section on Ophthalmology of the M.M.A. — Jay K. Osler, M.D., 74 Birch St., Bangor

Maine Radiological Society — John F. Gibbons, M.D., 22 Bramhall St., Portland  
Maine Chapter, American College of Surgeons — John F. Reynolds, M.D., 216 Main St., Waterville  
Maine Trauma Committee — John A. Woodcock, M.D., 35 Second St., Bangor  
Ear, Nose and Throat Group — John E. Whitworth, M.D., 336 Mt. Hope Ave., Bangor  
Maine Society of Pathologists — Franklin F. Ferguson, M.D., 22 Bramhall St., Portland

### Delegate and Alternate to AMA

January 1, 1963 to January 1, 1965

Delegate — Asa C. Adams, M.D., 68 Main St., Orono  
Alternate Delegate — Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville

January 1, 1965 to January 1, 1967

Delegate — Paul H. Pfeiffer, M.D., 14 Gilman St., Waterville  
Alternate Delegate — George W. Wood, III, M.D., 156 North Main St., Brewer



## News, Notes and Announcements

### Maine State Chairman Chosen for Deafness Research Foundation

George O. Cummings, Jr., M.D., well-known otolaryngologist, of Portland, has been appointed Maine State Chairman of the Deafness Research Foundation, it was recently announced by Gordon D. Hoople, M.D., Medical Adviser to the Foundation.

As Maine State Chairman, Dr. Cummings will act as spokesman for DRF at state and regional medical meetings, serve as liaison between the Foundation, the medical profession and the general public, and reply to inquiries about medical aspects of ear disorders addressed to the Foundation by people in Maine.

Dr. Cummings will also attempt to further DRF's educational program by soliciting the cooperation of all the news media of the state.

### Department of Health and Welfare Division of Maternal and Child Health Including Services for Crippled Children (By Appointment Only)

#### Orthopedic Clinics

Augusta—Augusta General Hospital

1:00 p.m.: Aug. 27

Bangor—Eastern Maine General Hospital

9:00 a.m. and 1:00 p.m.: July 23, Sept. 24

Half-day sessions 1:00 p.m.: Aug. 27

Fort Kent—Peoples Benevolent Hospital

10:00 a.m.: Sept. 9

Lewiston—Central Maine General Hospital

9:00 a.m.: Aug. 21, Sept. 18

Portland—Maine Medical Center

9:00 a.m.: Aug. 10, Sept. 14

(In conjunction with MMC)

Presque Isle—Arthur R. Gould Memorial Hospital

9:00 a.m. and 12:30 p.m.: Sept. 8

Rockland—Knox County Hospital

1:30 p.m.: Aug. 20

Rumford—Community Hospital

1:30 p.m.: Sept. 16

### State of Maine Board of Registration of Medicine Secretary — George E. Sullivan, M.D. Waterville, Maine

#### Physicians Licensed to Practice Medicine and Surgery in the State of Maine March 10-12, 1964

##### THROUGH EXAMINATION

Cesare G. Coletta, M.D., Mass. General Hospital, Boston, Mass.

Boruch Dodiuk, M.D., 35 Lafayette St., Yarmouth, Me.

Mariusz Ehrlich, M.D., 21 First St., Quincy, Mass.

Dincer Firat, M.D., Roswell Park Memorial Institute, Buffalo, N. Y.

Asteria M. Gabriel, M.D., Box C, Pownal, Me.

Walter M. Gerhold, M.D., 755 Fenimore St., Brooklyn, N. Y.

Samuel Lerner, M.D., Yale Univ. School of Medicine, New Haven, Conn.

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PHILIP BLINDER, M.D.  
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CARL J. HEDIN, M.D.  
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 Felix Scherzinger, M.D., 65 Bergen St., Newark, N. J.  
 Lionel R. Tardiff, M.D., 128 Sabattus St., Lewiston, Me.  
 Aleksandar T. Trajkovski, M.D., 91 Appleton Ave., Pittsfield, Mass.  
 Mehmet Kemal Yillar, M.D., Hudson River State Hospital, Poughkeepsie, N. Y.  
 Tadeo Zbyszcki, M.D., Bangor State Hospital, Bangor, Me.

#### THROUGH RECIPROCITY

Muriel Bigler, M.D., Pineland Hospital, Pownal, Me.

William M. Blackwell, M.D., Millinocket Community Hosp., Millinocket, Me.  
 Alan W. Boone, M.D., Presque Isle, Me.  
 Bernard Givertz, M.D., 131 Chadwick St., Portland, Me.  
 Joseph J. Hiebel, M.D., 106 Kiernan Dr., Rantoul, Ill.  
 Hans A. Holzwarth, M.D., 75 Bald Mtn. Dr., Bangor, Me.  
 John C. Van Pelt, M.D., 18 Glen Ave., Cape Elizabeth, Me.

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## Book Review

**Discoveries of Blood Circulation by Tibor Doby, M.D. Published by Abelard-Schuman, New York. Price \$6.50.**

This book traces the discovery of circulation of the blood from the time of Aristotle to Harvey. In the years from 400 to 200 B.C. with the work of Hippocrates, Aristotle and Erasistratus, beginnings were made of scientific medicine. Animal dissections were made as well as observations of humans being prepared for embalming. Valves in the heart were discovered and observations made on the arteries and veins. Between this time and the time of Galen, scientific medicine declined and it became fashionable to quote previous work without further experimentation. Galen, however, carried out many experiments. Leonardo De Vinci and Vesalius were also keen experimenters. For centuries, the works of Galen became

standard authority and it was considered almost heretical to disagree with these. Non-conformists had a rather hard time in those days since their usual fate was to be burned at the stake or to be drawn and quartered. Since dissection was forbidden, one of the ways to obtain anatomical information was to inspect the bodies of those who had been quartered. The last parts of the book deal with the discovery of the circulation by Harvey and Malpighi.

The book emphasizes the historical and cultural aspects during the lifetime of each of the subjects. This is necessary in order to understand the conditions under which these people worked. The book is well written and interesting and is also very readable. Its preparation obviously entailed a great deal of research into the life and times of the discoverers of the circulation.

C. V. NELSON, PH.D.  
 Portland, Maine

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*Speaker of the House of Delegates*, LINUS J. STITHAM, M.D., Dover-Foxcroft

<i>Councilors</i>	<i>Term Expires</i>	<i>Councilors</i>	<i>Term Expires</i>
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PAUL S. HILL, JR., M.D., Saco	1966	ASA C. ADAMS, M.D., Orono	1967
Second District		Immediate Past President	
CHARLES F. BRANCH, M.D., Lewiston	1966	ERNEST W. STEIN, M.D., Pittsfield	1965
Third District		Delegate to AMA	
EDWARD K. MORSE, M.D., Rockland	1965	ASA C. ADAMS, M.D., Orono	Jan. 1, 1965
Fourth District		Alternate Delegate to AMA	
GEORGE E. SULLIVAN, M.D., Fairfield, Chm.	1965	PAUL H. PFEIFFER, M.D., Waterville	Jan. 1, 1965
Fifth District		Executive Director	
JAMES C. BATES, M.D., Eastport	1967	DANIEL F. HANLEY, M.D., Brunswick	

*Secretary-Treasurer*, ESTHER M. KENNARD, Brunswick

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# The Journal of the Maine Medical Association

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## The Unnecessary Post-Partum Hemorrhage

RUDOLF G. WINKELBAUER, M.D.

The maternal death from unexpected catastrophic blood loss continues to rank among the leading causes of obstetrical mortalities. Modern blood bank facilities alone cannot provide the solution to the entire problem. Improving our alertness and technique in the conduct of labor may equally contribute to better maternal care.

Much has been learned in the practice of obstetrics and gynecology that is based strictly on palpation and its correct interpretation. It is very difficult to convey impressions of palpation, and consequently textbooks and other literature are handicapped in this respect. With some experience, however, we can make good use of our fingertips.

Blood loss is part of every delivery and it may be for this reason that the appraisal of actual blood loss is not taken seriously enough. We are trained to think of post-partum hemorrhage only after 600 cc. or more of blood have been lost. Unfortunately too little emphasis is placed on the circumstances and warning signs of impending massive bleeding. I have often wondered how we can accurately estimate blood loss by viewing the pan at the foot end of the delivery table. Certainly most of us cannot go beyond a general description of "little" or "more than average" or "a whole lot." By the same token many post-partum hemorrhages are never recognized in the delivery room and are not recorded statistically. Many seem to come to light only later when the ambulatory patient has fainted, showed pallor on the post-partum ward or anemia was found through routine laboratory work a day or so after the delivery. It would seem quite likely that the actual number of occurring post-partum hemorrhages is much higher than is generally believed. I am certain that much blood is wasted unnecessarily, not to speak of resultant blood transfusions with added morbidity and

mortality from mismatching, infection, and prolonged hospitalization.

Our attention should not end with the delivery of the fetus, particularly after the complicated forceps delivery. Ideally the newborn should be handed immediately to a competent nurse or the pediatrician if possible. The closest surveillance of the uterus following the completion of the second stage of labor is mandatory so that the third stage can be swiftly terminated in a physiological manner. The obstetrician must hold with one hand the fundus and not turn away from the mother while the newborn is cared for. Too much can happen too quickly. Especially the incompletely separated placenta, even without any external evidence of blood loss (the placenta lying across the contracted cervix), can amount to massive concealed uterine hemorrhage. A gentle but steady massage and palpation of the fundus would never let it get that far. We sometimes find ourselves in company of well meaning nurses who massage the fundus too thoroughly or strongly before the expulsion of the placenta. While the obstetrician is busy with the newborn the overenthusiastic nurse may inadvertently produce partial separation of the placenta resulting in brisk bleeding. Concomitant traumatization of the myometrium from rough handling of the fundus adds a considerable handicap to the uterine function. With the delivery of the placenta the scene is set for atonic bleeding, undoubtedly the most common type of post-partum bleeding. We may be too hasty in our efforts to repair the episiotomy and then leave the fundus unattended. It should be stressed that post-partum atony, this dreaded malfunction of the myometrium, can be suspected in its earliest stages before blood loss has manifested itself. Careful bimanual palpation at the beginning of the fourth stage of labor will detect pronounced flab-

business of the fundus and the lower uterine segment, that needs such prompt and undivided attention, as if hemorrhage had already occurred. In that case bimanual hugging of the uterus and gentle massage should be administered until strong contractions have taken over from whatever oxytocic has been administered. Then gentle massage will hopefully continue to maintain good tonus and will avert blood loss of significant degree. It is interesting to observe uterine behavior after expulsion of the placenta. An immediate strong contraction does not necessarily indicate that the same degree of tonicity will continue. Following every delivery, checks and rechecks of the fundus are worth the slight inconvenience and time.

I believe ether anesthesia is still much in use. Uterine atony following or during ether anesthesia is particular-

ly frequent. This seems to apply also to some extent to prolonged Trilene® inhalation. With this in mind an extra minute or two of preventive medicine can immediately correct the apparent insignificant degree of uterine softness that later on could snowball into atony par excellence. Are we not amiss in our judgment when to initiate treatment and must we wait for heavy blood loss to justify our efforts?

The atonic tendency is a palpable dysfunction. It can and should be recognized early, before the hemorrhagic deluge. It can be remedied in its earlier stages with oxytocics and massage alone, long before anemia and shock have compounded the threat to maternal life. One step ahead may make the difference.

Baribeau Drive, Brunswick, Maine

## Physical Basis for Restriction of Participation in Sports

A. A. SAVASTANO, M.D.\*

Competitive athletics are an important part of high school and college life in the United States today. This is principally due to the normal desire of a youth to stand out among others in the same age group and at the same time this desire affords the student an opportunity for a physical and emotional outlet. This indeed is a very happy state of affairs, keeping the mind and body of so many high school and college athletes occupied serves to reduce delinquency in this country.

Competition in all fields of sports should be encouraged; however, it is of utmost importance that the participants should not be given unrestricted permission to participate in sports without a careful medical checkup. Any imposed restriction should be carefully explained to the athlete and to his family whenever possible. Restrictions need not be total. A boy may be totally restricted from engaging in a given sport and yet be given unlimited permission to engage in other types of sports.

In addition to being physically fit, participants in sports should have the desire to engage in sports as well as to be willing to faithfully undertake a program of physical fitness activities, particularly during the summer. The latter is extremely important. One of the best ways to prevent athletic injuries is to have the athlete engage in progressive exercises during the summer to develop his muscle strength and endurance.

It is not enough to say that boys and girls of approximately the same age may engage in competitive sports. It is a common fact that boys in the same class are not of the same stage of physical development. One of the simplest methods of determining whether or not a boy or girl has reached maturity is to note the appearance of the pubic hair. As a general rule, when the pubic hair has extended to the inner thighs the individual has usually reached maturity and may therefore engage in sports with youngsters who have reached the same development but yet be of different ages.

Needless to say, a careful detailed medical history should be obtained from each prospective participant. Any history of previous serious medical disease or orthopedic deformity or problem should be probed.

Important items in the history which should be given particular consideration are a history of acute infection, hernia, weight loss, respiratory infections, rheumatic fever and cardiac lesions in addition to injuries and positive orthopedic problems.

The following conditions deserve special consideration in determining the athlete's fitness to participate in sports:

1. *Paired organs:* Any boy or girl who has lost one of any set of paired organs, should not be allowed to participate in high school or college contact sports. If a boy has lost one eye or one kidney or one testicle, he should automatically be disqualified. Some professional teams and some colleges and high schools make an exception to this; however, disqualification of such an athlete is in his best interest. I am told that one

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of the better-known professional football players, engaged in the sport for a considerable length of time although he only had one eye. I am also told that on one occasion he sustained a laceration just above the remaining eye which at the time caused him no little concern.

2. *Head injuries:* If a boy has a tendency to be knocked out, he should be eliminated from contact sports. These boys sustain the same injuries to their brain that the boxer sustains when he is knocked out. The final effect of repeated injury to the brain will be the same. This is the type of lad who could very well become the so-called "punch-drunk athlete." A few years ago we had one boy who was knocked out for just a few moments during football games on three successive Saturdays. We advised this boy to give up contact sports which he did. How many times does a boy have to be knocked out for a short period of time before you recommend that he be disqualified or be barred from engaging in contact sports? The answer would have to be a qualified one. The neurosurgeons feel that any boy who has been knocked out three times should be disqualified from contact sports; however, one lengthy period of unconsciousness should also result in medical disqualification.

3. *Neck Disturbances:* The long neck is more prone to injury than the short neck. Seventeen of the 1962 football deaths resulted from head and neck injuries. Boys with long necks should be discouraged from playing football.

A. *Ruptured disc:* It is best to eliminate a boy from contact sports, particularly football, if a ruptured cervical disc is suspected, the type of contact which is required in football affords him an excellent chance of making his condition worse.

B. *Wry neck:* I do not feel there to be any reason to bar these boys from contact sports.

4. *Acromioclavicular separations:* If these boys repeatedly experience additional pain when hit during contact sports, it is advisable for them to have the condition surgically repaired or withdraw from contact sports.

5. *Recurrent dislocation of the shoulder:* It may not be necessary to bar a boy who suffers from recurrent dislocation of the shoulder from contact sports. There is on the market a very excellent harness which reduces the incidence of re-dislocation during the actual participation in the sport. Surgical repair gives a high percentage of good results and restores these cases to full sports activity.

6. *Curvature of the spine:* There is no reason to restrict boys with lateral curvatures of the spine, dorsal roundbacks or sway-backs from contact sports, unless they are symptomatic. By the time the boy gets to college, the growth of the curvature has stopped. Even if it had not stopped, I do not believe that contact sports would make the condition worse.

7. *Diseases of the spine:* Athletes suffering from

tuberculosis or epiphysitis in the active stage should not be allowed to participate in contact sports. Active treatment is necessary in both of these conditions.

8. *Spondylolisthesis, sacralization, lumbarization, etc:* As far as sacralization and lumbarization are concerned, the restrictions from sports would have to be individualized. If there was no pain, I see no reason to restrict him from contact sports. If he did have pain, then, of course, the situation would be different. Spondylolisthesis, is an individual problem. Whether or not such a patient should be allowed to participate in sports would depend upon the degree of spondylolisthesis as well as on whether or not he had pain. If the athlete had a third or fourth degree spondylolisthesis, I would bar him from contact sports regardless of pain. If the boy had a first or second degree spondylolisthesis without pain, or other symptoms I would allow him to engage in contact sports.

9. *Old congenital dislocation of the hip, old Legg-Perthés disease or old slipped femoral epiphysis:* If these conditions existed but were not symptomatic I would not hesitate to allow these boys to play. X-ray evidence alone of these conditions would not cause me to bar these boys from sports. I would be the first one to admit that if one saw x-ray evidence of these conditions in the boys' hips, one could be sure that later on in life the boy would develop pain but I am of the opinion that engaging in contact sports would not hasten the onset of pain.

10. *Osgood-Schlatter's disease:* At the high school level one is concerned with Osgood-Schlatter's disease but one is not apt to be concerned at the college level. Whether or not the boy were allowed to engage in contact sports would have to depend upon the state of the disease. If the condition existed in a high school boy and were symptomatic, I believe that he should be eliminated on a temporary basis.

11. *Osteochondritis dissecans:* Restriction should be enforced until healing occurs or until a loose body is surgically removed.

12. *Torn semilunar cartilages or torn collateral ligaments of knees:* These of necessity must be dealt with on an individual basis. If a boy had a severely torn internal or external knee collateral ligament with gross instability, he should be eliminated from contact sports. If however, only mild instability was present, I would be inclined to allow these boys to continue athletics on condition that the knees were adequately externally supported during practice and during actual game competition and a program of progressive resistance exercises was followed. With internal semilunar cartilage cruciate ligament, injuries practically the same situation exists. If the knee repeatedly gave problems and these could not be prevented by adequate external support, then he should not be allowed to participate in contact sports.

13. *Recurrent dislocation of patella:* Restrict vigorous sports activities until surgically repaired.

14. *Infectious diseases:* When do you let a boy play after he has had an upper respiratory infection, infectious mononucleosis, pneumonia or any other condition which is due to infection. There are many ways of determining when the boy is ready to resume activities. First of all, he himself must feel strong, well and have the desire to return to contact sports. Then, of course, certain laboratory tests should be done and should have normal values. These should include a normal blood count, a normal sedimentation rate, a normal return of pulse rate after exercise is essential. The best test to determine whether or not an athlete is ready to return to active competition or active practice is the so-called "step test" as described by Gallagher and Brouha in the *Journal of Biology and Medicine* in 1942. This particular test involves the stepping on and off a platform 18 inches high thirty times a minute for four minutes. The heart rate is taken 30 seconds, 1 minute, 2 minutes and 3 minutes after the exercise. The rate of recovery is an excellent index in determining

the athlete's readiness to return to competition. The formula used is as follows:

$$\frac{\text{Duration of exercise in seconds} \times 100}{2 \times \text{sum of 3 pulse counts in recovery}}$$

The better the condition of the individual the higher the index. An index of 65 or less indicates that the person's condition is not satisfactory.

15. *Emotional problems:* If a boy complains of nervousness, of being tired, of generally not feeling well or lacking in enthusiasm, the trainer or coach or team physician should make a very serious effort to get at the bottom of such a complaint. In many cases the basic reason for the complaints is one dealing with finances, scholastic difficulties or problems with cupid. Trainers, coaches and team physicians enjoy a key position in dealing with the emotionally-disturbed boy. These boys often look upon the trainer, team physician or coach as their father confessor or friend and are, therefore, very apt to unfold their problems to him.

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## Venous Obstruction of the Antecubital Fossa

ROWLAND B. FRENCH, M.D.

Much has been written about edema of the arm but largely having to do with axillary surgery and axillary vein thrombosis.<sup>1,2,3</sup> Volkmann's ischemic paralysis has followed severe injuries to the antecubital fossa not necessarily associated with fracture.<sup>4,5,6</sup> However, these appear to have been largely associated with extreme tenderness and swelling over the region of obstruction.

### CASE REPORT

The patient, a seventeen-year-old white female, was seen with a chief complaint of pain in the right elbow antecubital fossa of two to three days duration, associated with swelling and numb sensation of the right hand and forearm.

Examination revealed the right hand to be moderately swollen and moderately blue. The swelling extended approximately to, but not including, the antecubital fossa with maximum swelling in the hand. A good radial pulse was present and the hand was warm. There was tenderness over the mid portion of the antecubital fossa but no evidence of swelling of the elbow region.

Physical examination revealed weight 140 pounds; blood pressure 115/80; heart not enlarged, regular, lungs to be clear; Hgb 14.1 gm per 100 cc blood; Urine, Reac., acid, Sugar, negative, Albumin, negative, Microscopic examination, negative.

There was no definite explanation for the cyanosis and swelling of the hand and the tenderness in the mid-antecubital space. However, the patient did give a history of carrying books approximately three days earlier with the possibility of pressure on the antecubital space, followed by tenderness of the space and swelling of the hand. The patient was treated by elevation and rest of the extremity with gradual disappearance

of the swelling and cyanosis. The swelling largely disappeared approximately five days from onset, but she still retained a numb feeling of the forearm and tenderness over the mid-antecubital fossa. This disappeared over a ten-day period, although the patient complained of occasional soreness of this area for some weeks more. Chest X-Ray three months later was reported normal.

### COMMENT

The deep veins of the antecubital fossa are arranged in pairs accompanying the brachial artery.<sup>7</sup> In the case reported here, the superficial veins, the basilic and cephalic, apparently did not accommodate enough blood flow to avoid swelling. It was felt that the deep fascia and bicipital aponeurosis was not the source of obstruction as no swelling or tenseness was present in the antecubital fossa as there would have been if the brachialis muscle or antecubital contents were swollen. There was only tenderness in the mid-fossa region. The cause of the swelling and cyanosis of the hand and lower forearm was believed due to venous obstruction. However, the lack of swelling in the antecubital space made the usual explanation of venous compression less likely and it is postulated that venous spasm or even venous thrombosis may have occurred. Five year follow-up has failed to reveal any evidence of recurrence or further difficulty.

*Continued on Page 151*



# Attitudes of College Preparatory Seniors in York County, Maine Toward Medicine as a Career Choice

EDWARD J. DURNALL\* and JOHN C. MYER, M.D.\*\*

Non-urban states such as Maine face a critical shortage of practicing physicians. The present ratio of physicians to population on a national basis is 133 doctors for each unit of 100,000 population. The Maine picture is 84 physicians for every 100,000 people. Young doctors are not coming into the State and those few that do enter practice in Maine tend to concentrate in the larger, more urbanized centers. If larger numbers of intelligent and well-motivated young people are to be drawn into the medical profession, it is important that a realistic appraisal be made of the circumstances that seem to limit the supply of students today. Clearer understanding of present obstacles to medical education is essential as a basis for devising ways to encourage a larger proportion of able young people to seek entrance to medical schools. No analysis of the views and attitudes of high school students towards medicine as a career choice has been made in the State of Maine. This study was conducted to investigate the perception that young people have of medicine as a career choice.

The study was conducted with high school seniors in York County who were completing a college preparatory course as defined by their high schools. An attitude scale of 100 items was completed by 310 high school seniors enrolled in York County schools. A total of 294 usable cases composed of 159 males and 135 females constituted the sample studied. The attitude inventory developed consisted of 100 items which were subdivided into six attitude scales and a separate validating scale. The six scales utilized were as follows:

- Scale 1: self-image in terms of the ability to complete a training program of some difficulty
- Scale 2: expense involved in becoming a physician
- Scale 3: length of time involved in becoming a physician
- Scale 4: interest in the medical profession
- Scale 5: unfavorable image of the physician
- Scale 6: favorable image of the physician

An analysis was first made of the information supplied by the students under the "probable choice of college major" category. As a result of this analysis, nine categories were chosen which were judged to be sufficiently inclusive to cover all of the areas of vocational choice expressed by the respondents. Table I presents these categories with the number of respondents in each.

The first hypothesis examined was that there would be a positive relationship between the student's image of his own capabilities and his plans for further edu-

Table 1

Probable Choice of College Major			
Probable college major		N male	N female
I	not going to college	11	21
II	undecided	19	12
III	education, teaching	7	20
IV	business, secretarial, home economics	17	16
V	humanities	10	21
VI	the social sciences	24	11
VII	engineering	31	0
VIII	nursing	0	22
IX	science, mathematics pre-medical	40	12

Table 2

Scores on Scale 1 by Probable Choice of College Major				
Probable college major (See Table I)	Male		Female	
	mean	S.D.	mean	S.D.
I	7.00	3.88	4.81	4.51
II	4.95	4.69	6.00	4.25
III	8.71	5.09	6.95	5.66
IV	7.52	4.92	6.06	5.75
V	3.80	3.51	4.14	4.60
VI	4.12	4.05	5.36	4.91
VII	4.06	4.09	—	—
VIII	—	—	2.86	3.40
IX	1.32	2.16	2.50	4.27

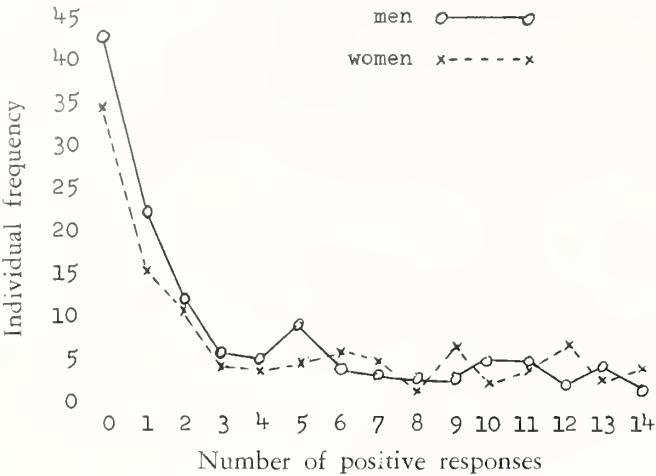
cation. Table 2 presents these data with a high score indicative of a poor self-image and conversely a low score representing a favorable self-image.

It was clear that the least favorable self-images in terms of ability to complete a training program of some difficulty were held by students planning on careers in education and business, categories III and IV as shown in Table 2. The most favorable self-images were held by students planning college majors in categories IX and VIII; namely, science and nursing. Statistically significant differences were found between the mean scores on the self-image scale and the student's probable choice of college major.

A second finding relative to the self-image scale was that those students with favorable self-images were more inclined to be interested in medicine as a career than were those students with poorer self-images. A correlation of .303 which was significant at the .01 level

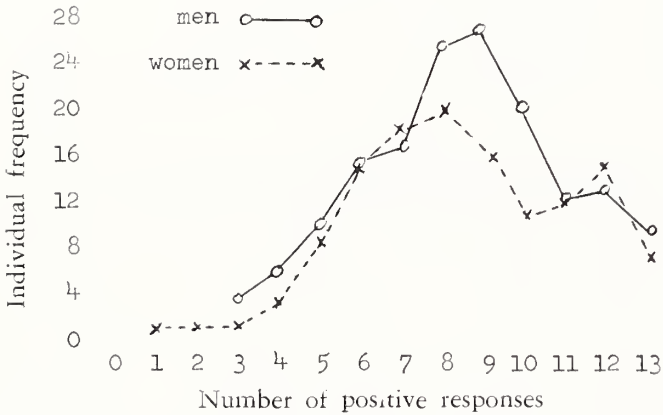
\*Dean, Nasson College, Springvale, Maine.  
\*\*College Physician, Nasson College, Springvale, Maine.

FIGURE 1



Frequency polygon for scale 1 scores.  
Self-image in terms of ability to complete a training program of some difficulty

FIGURE 2



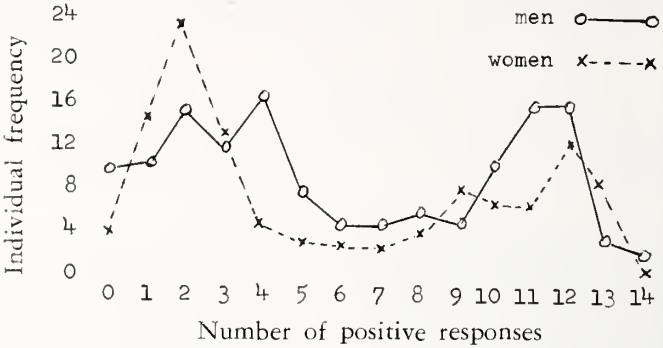
Frequency polygon for scale 2 scores.  
Expense involved in becoming a physician

of confidence was obtained between the scores on the self-image scale and the interest in medicine scale.

Figures 1-6 present frequency polygons summarizing the responses of the men and women on Scales 1-6. One notable finding from these data is the very favorable view the students held of the physician. While it may be true that the number of students entering medical school declined substantially since World War II, it appears that the students surveyed in York County, Maine, look upon the physician as an almost perfect career symbol.

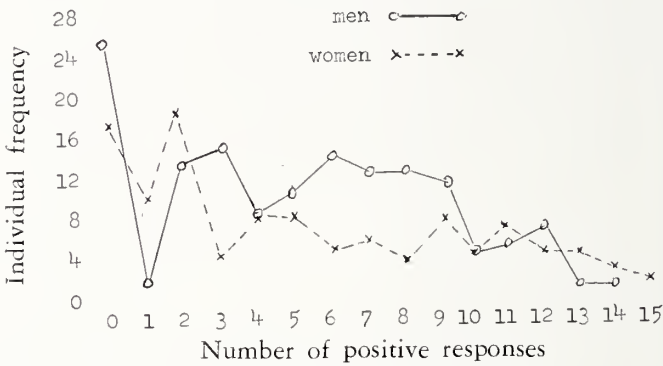
The data were further analyzed through more sophisticated techniques including analysis of variance using a 2x2x2 factorial design, a 2x2 analysis of variance, and chi-square analysis. Those students who expressed an interest in medicine did not appear to consider the time factor relevant. A more detailed analysis of a subgroup of students with the highest self-image and strongest interest in medicine indicated that those students who would appear to be the most likely candidates for the medical profession were less concerned

FIGURE 3



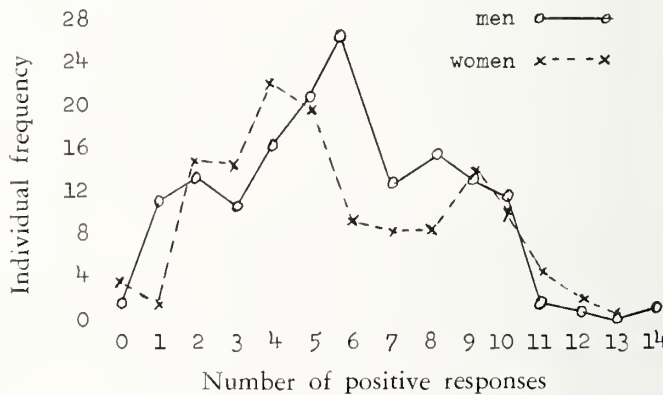
Frequency polygon for scale 3 scores.  
Length of time involved in becoming a physician

FIGURE 4



Frequency polygon for scale 4 scores.  
Interest in the medical profession

FIGURE 5



Frequency polygon for scale 5 scores.  
Unfavorable image of the physician

with the time involved than were their fellow students who might be considered less likely candidates for the profession.

The students as a whole, as well as those most favorably disposed toward medicine as a career, were strongly in favor of government sponsored scholarships for medical school. While those students who were disposed toward medicine were concerned with the economic problems, they did not seem to feel that these were insurmountable problems for them personally. However,



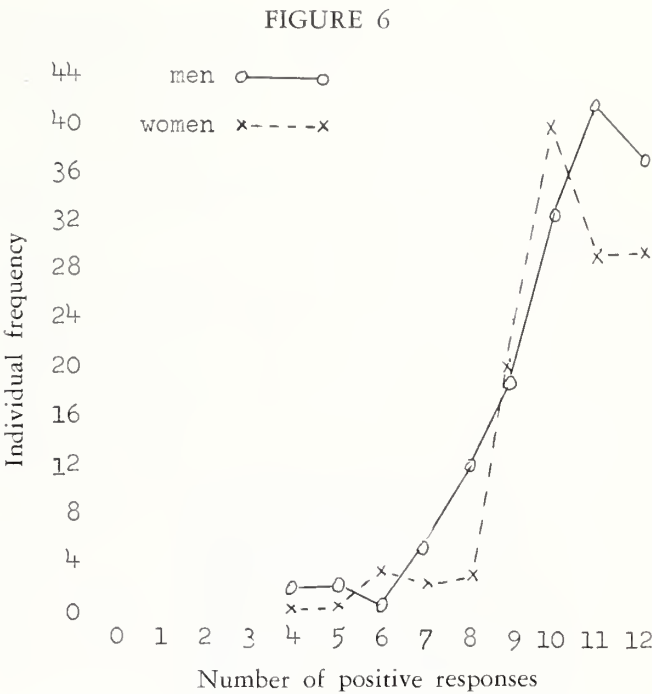
factors may be operating so that those who did feel that the economic problems were beyond their capacity had already eliminated medicine as a career in their minds and therefore expressed lack of interest in medicine.

There were several significant differences between the responses of the men and women. Women were more likely to feel that the government should establish scholarships whereas the men were more likely to merely recognize the expense involved in becoming a physician. There was also a sizeable group of women who felt that the only problem involved in becoming a physician was the time involved, whereas the men were more general in their views of the time limitation. Women who expressed lack of interest in medicine were more likely to mention specific reasons such as lack of drive, while the men merely stated a general lack of interest. So far as a negative image of the physician is concerned, the women criticized the over-emphasis on research, high fees, and lack of interest in the patient while the men saw science as being more important and they did not like the physician's irregular schedule.

IMPLICATIONS

It would seem that attitudes toward medicine as a career are fairly well formulated by the time a student completes high school. A major factor contributing to these attitudes is the self-image so far as the student's ability to complete an educational program of some difficulty. A selective process seems to be operating so that those students who view their abilities as being somewhat limited tend to plan on careers in vocations which are usually not considered intellectually demanding while those students with favorable self-images select more rigorous disciplines.

In order to effectively interest more young people in medicine as a career, an approach tailored to the specific group most likely to consider medicine as a career should be developed. Only those who hold favorable views of their abilities should be approached and a campaign should be conducted to point out that



Frequency polygon for scale 6 scores.  
Favorable image of the physician

medicine is as important as research and basic science and in fact offers many opportunities to those interested in these areas. The lack of set hours of work in medicine obviously only applies to certain physicians and efforts should be made to point out to young men that many doctors have regular schedules made possible through group practice, specialization and research positions. The approach to young women with favorable self-images should emphasize the fact that the government has now established scholarships for medical school and that the time involved in training may be reduced in certain cases by early admission to medical school. The fact that other professions are lengthening their training programs should also be a strong argument to use with this group. The human side of medicine with emphasis on the doctor-patient relationship should be pushed in any campaign to interest women in medicine as a career.

VENOUS OBSTRUCTION OF THE ANTECUBITAL FOSSA — Continued from Page 148

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# President's Address\*

ERNEST W. STEIN, M.D.\*\*

Mr. Speaker; members of the Council; Delegates and honored guests:

As I stand before you today, with mixed emotions, I view the future with awe and horror. Today, Flag Day, I observe the anniversary of my birth date, and look to a future of tottering senility. Today I sing the Swan Song of my office in the State Medical Society and look forward to uncertain usefulness; and today I am being supplanted as the leading physician of my community by a much younger, better trained, more scientific man — just as I took over from the much older, endeared family general practitioner when I moved to town years ago. Perhaps the most consoling thought arises, as the words of the ballad made famous by that great soldier General MacArthur, even unto death itself, keep reverberating in my ears — "Old soldiers never die, they just fade away."

The Doctor of today is midway between two eras, a rapidly changing scene. For generations the American public has idolized the American physician, but this ardor has rapidly cooled. The paradox of it is that never before in history has the patient been given such good care by such highly trained and stringently regulated physicians. Never before have Americans lived so long, enjoyed such good health and been so free of crippling diseases. Modern day medical miracles are the every day tool of the M.D. and he is using them with astonishing success. Never before has the patient paid so little per unit of health and gotten so much from his physician. Yet, never before in the history of American medicine has the American physician been the subject of so much complaint and criticism. Despite his success with diagnostic drugs and techniques, there is an undercurrent of discontent about him and a mental malaise that shows itself in recurrent complaints, the constant threat of previously unheard-of malpractice suits and barbs from government, labor unions, the "Golden Year" groups, etc. Beneath all these complaints, with many people, runs a subcurrent of desire for the golden past, for the warmth and comfort of the "horse and buggy" doctor. In actuality, these days were hardly golden as far as medical science was concerned, but fact rarely interferes with a legend, and the modern American doctor still works in the shadow of kindly men now dead.

Medicine may be looked upon as a balance, with the Art of Medicine in the bygone days far outweighing the Science. The newer generation appears to stress the Science of Medicine and Science far outweighs the Art. It is an incontrovertible fact that the Science of Medicine will continue to grow by leaps and bounds in the future to the point that the Art of Medicine may be entirely lost

and forgotten. The old doctor, for all his warmth and the time he could spend with his patients, is a vanishing breed. We can't stop his disappearance — it's inevitable. Even the public doesn't really want him anymore, not in terms of his medical knowledge. Without a doubt it is specialization that has made American Medicine as great as it is today. The role of the General Practitioner has been greatly minimized, mainly as a result of the tremendous burgeoning knowledge. Advances in all fields of medical research and care relegate the family physician to the role of an assorting and distributing bureau in a great percentage of cases.

What have we to look forward to in the future? — various socio-economic reforms resulting in loss of personal initiative, even total government controlled medical care, hospitalization and research — the utter loss of warmth and personality in physician-patient relationships — a gradually diminishing stature for the community physician — or even a prohibitive economic cost, variously estimated at upwards of \$70,000.00 for a medical education in a specialty from high school until the day the doctor hangs out his shingle.

Times have changed; science has changed; the patient has changed; and so has the physician. And with these changes, of course, the image of the American doctor has changed. The doctors innate independence is not helping to form a fonder image, and he is unlikely to change in this respect. The patient will not become less educated, less demanding or less critical. In addition, Americans — despite the cries of organized medicine — are beginning to feel that medical care is a "right" and not much different from such established services as education, fire protection and public roads, regardless of who pays for it or how.

The American Medical profession is in a battle for its veritable life, independence and individuality and as blasé as each of us is with regard to medical politics, medical public relations and concern for our public image, it behooves each and every one of us to scrutinize our predicament and then pitch in individually and collectively in an all out effort to stem the adverse public opinion which has been sweeping us out of first place in popularity contests throughout the country.

In traveling from Kittery to Ft. Kent and Calais to Bethel, I was appalled at the apathy manifest by physicians and Medical Societies throughout the State relative to matters directly pertinent to the medical profession, locally, statewide and nationally. My parting plea to you is that you all assume active roles in your society and better familiarize yourself and your colleagues with medical matters that can influence the future of yourselves, your families and future generations. Only by such a course can we assume our rightful place in society today and carry the proud heritage which has always been rightfully that of the medical profession.

\*Presented at the 1964 annual session of the Maine Medical Association.

\*\*President, Maine Medical Association, 1963-1964.



Elected at the 111th Annual Session  
of the Maine Medical Association

Rockland, Maine

June 14, 15, 16, 1964

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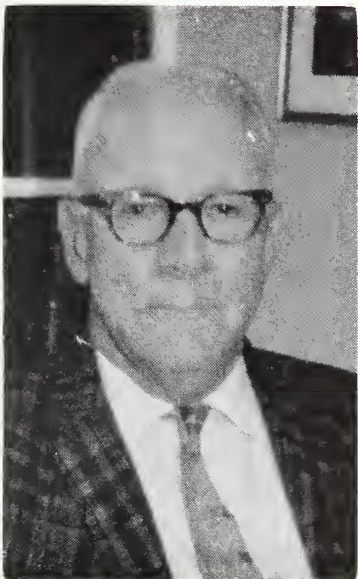
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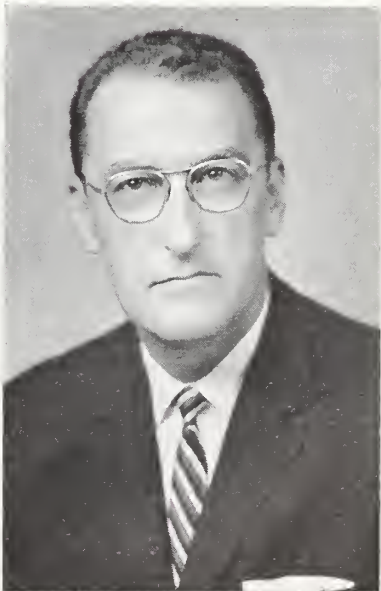
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DR. ADAMS



DR. SULLIVAN

# Report of Delegate to AMA House of Delegates Annual Meeting, San Francisco, California, June 21-25, 1964

ASA C. ADAMS, M.D.

This summary is presented by your Maine Medical Association delegate to the San Francisco meeting. It covers what I believe to be the more important subjects dealt with by the House of Delegates at this meeting.

If any member is particularly interested in any subject not mentioned here, I would be glad to discuss it further.

Tobacco and health, human rights, physician-hospital relations, continuing medical education, the cost of medical care, and federal subsidization of prepayment plans and health insurance companies were among the major subjects acted upon by the House of Delegates at the American Medical Association's 113th annual convention held June 21-25 in San Francisco.

Dr. Donovan F. Ward of Dubuque, Iowa, vice president of the Association, was named President-Elect of the Association. He will become President at the June, 1965 annual convention in New York City, succeeding Dr. Norman A. Welch of Boston, who was installed at the inaugural ceremony in San Francisco.

## TOBACCO AND HEALTH

The House approved a strong stand on tobacco and health by calling cigaret smoking a "serious health hazard." This action was taken after the reference committee on Public Health and Occupational Health considered 10 resolutions and a Board of Trustees report on the subject and heard considerable testimony.

In adopting a four-point reference committee report, the House said "the American Medical Association is on record and does recognize a significant relationship between cigaret smoking and the incidence of lung cancer and certain other diseases." It urged that programs be developed to disseminate vital health education material on the hazards of smoking to all age groups through all means of communication. The House also recognized the contribution of the Surgeon General's Committee in its comprehensive report. And it emphasized that a joint committee of the AMA and the National Education Association already has adopted a resolution urging elementary and secondary schools to include programs on smoking and health in their health education curricula.

The House further recommended that the AMA pamphlet, "Smoking Facts You Should Know," should be modified "in the light of accumulating knowledge."

"The Board of Directors of AMA-ERF and the Board of Trustees of the AMA were clearly aware of the possibility of criticism in accepting this grant of 10 million dollars from several tobacco companies. But against that possibility they weighed the potential benefits to the public who will continue to smoke and concluded that the risk was insignificant by comparison. The only hope of minimizing the hazards of smoking lies in research which points to the course that the AMA as well as others must take."

## HUMAN RIGHTS

On the major issue of human rights the House declared itself "unalterably opposed to the denial of membership, privileges and responsibilities in county medical societies and state medical associations to any duly, licensed physician because of race, color, religion, ethnic affiliation, or national origin."

This action was taken after the reference committee had heard a detailed discussion and had considered four resolutions on the subject. In addition, the House called "upon all state medical associations, all component societies, and all individual members of the AMA to exert every effort to end every instance in which equal rights, privileges and responsibilities are denied."

The House also accepted a report from the Board on the liaison committees of the AMA and the National Medical Association. This report reviewed the history of the committees and noted that "great progress has been made voluntarily. More progress can reasonably be expected in the immediate future, especially if the committees are permitted to continue on a constructive, cooperative basis. This requires effort, but more importantly, good will and the desire to eliminate problems."

## PHYSICIAN — HOSPITAL RELATIONS

Conclusions and recommendations in a significant and extensive report on physician-hospital relations were adopted by the House. Prepared by the Council on Medical Service's Committee on Medical Facilities, the report stresses "the imperative need for the medical profession to assume responsibility for the quality, continuity, and availability of professional services and for the coordination of these services with the other essential supportive aspects of health care."

The report's recommendations are designed to serve as guidelines for physicians in meeting the problems involved in the changing patterns of care such as: appointment of salaried chiefs of staff, appointment of salaried heads of clinical departments, appointment of salaried directors of medical education, employment of salaried physicians for outpatient and emergency departments, use of salaried physicians to provide care ordinarily provided by interns and residents, and utilization of closed panel prepayment medical care programs by hospitals.

The report also includes a review of the development of AMA's policy on physician-hospital relations, a study of the relation of policy to actual practice, and an investigation of the factors influencing change — including graduate education, medical finance, expansion of hospital functions and regulation of medical care.

## CONTINUING MEDICAL EDUCATION

Authorization was made by the House to establish an



AMA sponsored survey and accreditation program in continuing medical education. In the program, attention will be concentrated on institutions and organizations offering courses rather than on individual courses, and appraisal of an institution's or organization's program will be carried out only at its request.

Eventually, approved institutions or organizations will be so designated in the council's annual lists of "Continuing Education Courses for Physicians," and when all institutions which list their courses have had the opportunity to be considered for approval, only courses of approved institutions and organizations will be included in the annual list. Programs will be surveyed by a Review Committee on Continuing Medical Education.

#### COST OF MEDICAL CARE

A four volume report of the AMA Commission on the Cost of Medical Care was received by the delegates, and the House concurred with the Board of Trustees that the conclusions and recommendations of the Commission will be studied and a report will be made to the House for its consideration at the 1964 Clinical Convention.

The four volumes include a general report on factors involved in medical care costs, a full report on "Professional Review Mechanisms," another on "Significant Medical Advances," and one on "Changing Patterns of Hospital Care."

In its report the Board said that the Commission "is aware that its efforts will not result in a magic reduction in the price of medical and hospital services. It does believe, however, that its study has produced a considerable amount of new and relevant information which will serve as a basis for better understanding by the public and the medical profession of this complex subject."

Reaffirmed the AMA policy favoring federal grants for "bricks and mortar" — funds for construction and renovation of medical schools, hospitals, related institutions, and mental health centers — but urged that the "advantages and desirability of multiple source financing be kept clearly in mind." The House also was informed by the Board that it is appointing a commission to conduct a broad study of the role of federal support of medical research.

#### OTHER ACTIONS

The House went on record as opposing federal subsidization of prepayment plans and health insurance companies, and it asked for an AMA study of the development of state programs which utilize prepayment plans or health insurance companies in the implementation of state programs of medical aid to the aging under the Kerr-Mills law.

A proposal to poll all AMA members concerning compulsory Social Security for self-employed physicians was rejected by the House. In addition, the House concurred with the reference committee in opposing polls of the membership on issues of "great or even moderate importance" because the House members express the majority sentiments of their constituents on all questions coming before the House.

The House also approved the creation of the Section on Allergy on recommendation of the Board of Trustees.

Approved a comprehensive inquiry of the causative factors for the sharp increase in syphilis and gonorrhea and urged the AMA to "take leadership in educational and research measures designed to control and eliminate syphilis."

Agreed to a national conference on areawide planning of hospitals and related health facilities, to be sponsored under the auspices of the AMA.

Agreed to continue and broaden studies on the problems of unwed mothers, illegitimacy and other related matters and to develop positive preventive programs.

Supported a positive statement on protecting children against physical abuse and called for legislative guidelines to the state relative to legislation on this matter.

Asked the Board of Trustees to investigate establishment of a wire communication system between AMA headquarters in Chicago and offices of state medical associations.

Referred to the Council on Medical Service a resolution condemning the practice by some hospitals of adopting constitutions which deny staff privileges to physicians not eligible or certified by specialty bodies or societies.

Agreed with the Board that a forum for representatives of national medical specialty societies and the American Academy of General Practice be held on November 1, 1964 in Chicago.

Recommended that the Board of Trustees use the talents of Dr. Edward R. Annis, immediate past president, and other qualified spokesmen for medicine with appropriate remuneration.

Recommended that the Board of Trustees approve the establishment of an ad hoc study on family practice as proposed by the Council on Medical Education.

#### ELECTION OF OFFICERS

In addition to Dr. Ward, the new president-elect, the following officers were named:

Dr. Carlton Wertz of Buffalo, vice president; Dr. Milford O. Rouse of Dallas, speaker of the House; and Dr. Walter C. Borneimeier of Chicago, vice speaker.

Dr. Robert C. Long of Louisville was reelected to the Board of Trustees for a three-year term, and Dr. Alvin J. Ingram of Memphis was elected to a three-year term.

Nominated and elected to the Judicial Council was Dr. Charles C. Smeltzer of Knoxville, Tenn. Named to the Council on Medical Education were Dr. William P. Longmire of Los Angeles, and Dr. William A. Soderman of Philadelphia.

Elected to the Council on Medical Service was Dr. John Rumsey.

In voting on the resolution that all members of the AMA be polled in regard to compulsory Social Security, the vote appeared to be closer than at any other time in the past. I believe that most delegates whose states had voted for Social Security, including your delegate, voted in favor of the motion, but it lost by a definite majority.

In Dr. Edward Annis' address to the delegates, he

*Continued on Page XV*



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## Summary of Rabies Outbreak in Maine

CHARLES H. OKEY, PH.D.\*

The current outbreak of rabies in western Maine began with the finding of a rabid fox in Rangeley on March 18, 1964. Since that time, a total of twenty-two animals have yielded rabies virus on testing in the Diagnostic Laboratory. Distribution was as follows: sixteen foxes, three skunks, one raccoon and two cows. The latter two animals are the only domestic species found to be rabid among a number of such animals examined in the laboratory.

As of late July, the outbreak was confined to the central Franklin County towns of Byron and Roxbury and the Oxford County towns of Lang, Rangeley, Madrid and Phillips and Rangeley and Dallas Plantations. The outbreak represents a spread from rabid wild animals known to exist in adjoining New Hampshire. Rabies there had been introduced earlier from Quebec Province after a slow eastward move of the disease from the western provinces beginning more than fifteen years ago. Northern New York State, Vermont and New Hampshire all experienced rabies in the past two years for the first time in thirty years. There have been no human cases in these states; however, a Canadian girl died of rabies after exposure by a skunk just north of the New York border.

An outbreak involving three foxes along the Maine-Quebec border in the fall and winter of 1962-63 was controlled by a fox poisoning campaign and no further cases have been found in the area. During 1962 Quebec health authorities reported approximately fifty cases of rabies with about half of the cases in domestic animals.

In September 1963 a rabid cat was found in Guilford. No other rabid animals have been seen, although a number of wild and domestic animals from the area were examined in subsequent months. Rabies virus exists in the area at an extremely low level or is imported by migratory bats. Bats examined in the Massachusetts laboratory indicate an infection rate for rabies in bats at 2%.

The Bureau of Health in cooperation with the Departments of Agriculture and Inland Fish and Game

developed a rabies control program this spring after the first rabid fox was found in Rangeley. Regulations to control stray dogs and a supplementary licensure system for dogs was put into effect. The licensure was based on mandatory immunization. Clinics for dog and cat immunization were held in the communities affected. Poison baits were distributed under controlled conditions over the areas known to have rabies plus a buffer zone around the critical area. The buffer zone was utilized to remove foxes thought to be the principal vectors for the virus.

Two health educators have been assigned by the department to the poisoning teams. They are responsible for contacting town and other local officials to inform and expedite the poisoning operations, to encourage enforcement of regulations, to encourage reporting of fox sightings, to explain the control program, to observe the degree of enforcement of regulations, and to do whatever other educational or informational work that may contribute to the effectiveness of the program. They will also contact individual residents in areas to be poisoned to warn them of the poisoning, to inform, and to reassure. They will coordinate their work with the poison teams through conferences, and directions received from the local biologist in charge.

General information regarding rabies and the poison campaign has been made available to residents of the area. Rabies hyperimmune serum and duck embryo vaccine has been distributed to hospitals and physicians' offices. The department will supply whatever educational material that may be needed in the above respect. It will also be glad to conduct whatever kinds of public meetings in the area that may contribute to the effectiveness of the control work.

The rabies vaccine available from the Bureau of Health is the duck embryo type. Lacking significant amounts of neural tissue, the vaccine is administered with a minimal hazard of the allergic reactions associated with older vaccines containing nervous tissue. Only two marked reactions to the duck embryo vaccine have been reported from a total of more than 50,000 individuals receiving the preparation. Neither of these reactions resulted in permanent damage.

\*Director, Diagnostic Laboratory



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## PRO-BANTHINE (propantheline bromide) Assures Authoritative Anticholinergic Control in Gastrointestinal Dysfunctions

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Early investigations showed that Pro-Banthine (propantheline bromide) reduces motility and acid secretion and may be used in a wide range of dosage, to bring prompt, positive anticholinergic benefits to patients with peptic ulcer, spastic colon, pylorospasm and related gastrointestinal dysfunctions.

Recent evaluations sustain these earlier judgments. In a current authoritative assessment based mainly on the factors of potency, superiority to atropine, clinical experience and physiologic study, Steinberg and Almy<sup>2</sup> select as the first two preferred anticholinergic drugs, methantheline [Banthine] and propantheline [Pro-Banthine].

The name Pro-Banthine (propantheline bromide) sets a stamp of therapeutic authority on any anticholinergic prescription.

*Side Effects and Precautions*—Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. The drug is contraindicated in patients with glaucoma or severe cardiac disease.

*Dosage*—The usual adult dosage is one tablet of 15 mg. with meals and two at bedtime; this amount may be doubled or tripled for patients with severe conditions. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

### SEARLE

Chicago, Illinois 60680

*Research in the Service of Medicine*

1. Roach, T. C.: Therapy of Peptic Ulcer, J. Louisiana Med. Soc. 115:136-139 (April) 1963.
2. Steinberg, H., and Almy, T. P., Drugs for Gastrointestinal Disturbances, Chapter 21, in Modell, W. (editor): Drugs of Choice—1964-1965, St. Louis, The C. V. Mosby Company, 1964, p. 343.

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## *Maine Heart Association Notes*



### **"Environmental Factors in Ischemic Heart Disease"**

"As a group, the Seventh-day Adventists may provide the basis for a comparison of the effects of an atypical vs a typical American environment upon ischemic heart disease. The members of this sect use much less meat and fat of animal origin than the average American. They do not use beverages containing caffeine and only rarely use tobacco or alcohol. . . . reported that hospital admissions for coronary artery disease were approximately 40% less among Seventh-day Adventist men and 15% less among Seventh-day Adventist women than among men and women from typical American environments. The serum cholesterol level of adult Seventh-day Adventists . . . was found to be substantially lower than that in age-matched control groups in New York City. . . . Dietary factors appear to be involved; in the diet of the Seventh-day Adventists, only 30% of the calories consist in fat and 55% are in the form of carbohydrates. . . . Abstinence from tobacco and alcohol, as well as the low intake of caffeinated beverages among this sect, may also be an important factor to explain the serum lipid differences.

". . . An association between coronary artery disease and changing patterns of serum cholesterol levels with increasing age is suggested by the fact that both the peak incidence of coronary heart disease and the sharp increase in serum total cholesterol occurred 10 to 15 years later in the male Seventh-day Adventists than among other American men. A relationship between smoking habits and ischemic heart disease is also suggested by a study of both groups. . . .

"The striking cardiological differences between the Seventh-day Adventists and the general population may indicate a fundamental role of environmental factors in ischemic heart disease. Further study may provide understanding of the relative roles of nonsmoking and teetotaling habits and a largely vegetation diet."

---

Reference: Journal of the American Medical Association, Editorial. Volume 188, No. 11, page 997, 1964.

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### **Maine Heart Association Annual Meeting**

The fifteenth Annual Meeting of the Maine Heart Association will be held at the Holiday Inn, Auburn, Maine, Wednesday, September 23, 1964. Featured speaker for the meeting will be William C. Elliot, M.D., Assistant in Medicine, Peter Bent Brigham Hospital, Boston, and Research Fellow in Medicine, Harvard Medical School. His subject will be "Modern Methods of Diagnosis and Treatment of Coronary Occlusion and Insufficiency." For reservations, write Maine Heart Association, 116 State Street, Augusta, Maine.



## Announcements

### Seminar on the Treatment of Complete Heart Block

On October 10 and 11, 1964, a Seminar on the Treatment of Complete Heart Block, sponsored by the Vermont Heart Association and the University of Vermont College of Medicine will be held at the DeGoesbriand Memorial Hospital, Burlington, Vermont. In addition to the regular members of this College, Samuel Bellet, M.D. (or co-worker), William M. Chardack, M.D., Gordon K. Moe, M.D. and Paul M. Zoll, M.D. have agreed to participate as guest speakers.

Further information can be obtained from Eugene Lepeschkin, M.D., Cardiovascular Research Unit, DeGoesbriand Memorial Hospital, Burlington, Vermont.

### Trooper Black Fund

Each day we receive a number of inquiries from people who wish to participate in the formation of some type of tangible memorial for Trooper Charles Clinton Black. As you know, Trooper Black was gunned down while attempting to apprehend two bank robbers in South Berwick on July 9, 1964.

Trooper Black left a wife and two children, Clinton 5, and William 2, and his widow is expecting a third child.

We are happy to report that interested citizens have organized a "Trooper Black Fund." This will be a trust fund to provide for the health, welfare and education of the slain trooper's children.

Governor and Mrs. John H. Reed will serve as co-chairmen of this trust fund. Persons interested may forward checks for the "Trooper Black Fund" to Governor and Mrs. Reed, State House, Augusta, Maine, or to any bank, trust company, or savings and loan association in Maine.

COLONEL ROBERT MARX  
*Chief, Maine State Police*

### REPORT OF DELEGATE TO AMA HOUSE OF DELEGATES ANNUAL MEETING

*Continued from Page 155*

emphasized that some quicker means of communication between the State Societies and the AMA should be established, possibly teletype. This would call for an increase in dues, and he proposed that the dues be increased to \$100 in three steps. This matter was referred to the Trustees and will be discussed at the meeting in Miami in December.

This San Francisco meeting was attended by 14,229 physicians and Maine was represented by Dr. Thomas Martin, president of the Maine Medical Association; Dr. Clyde Swett, delegate from the Surgical Section; Dr. Paul Pfeiffer, alternate delegate; and myself as delegate.

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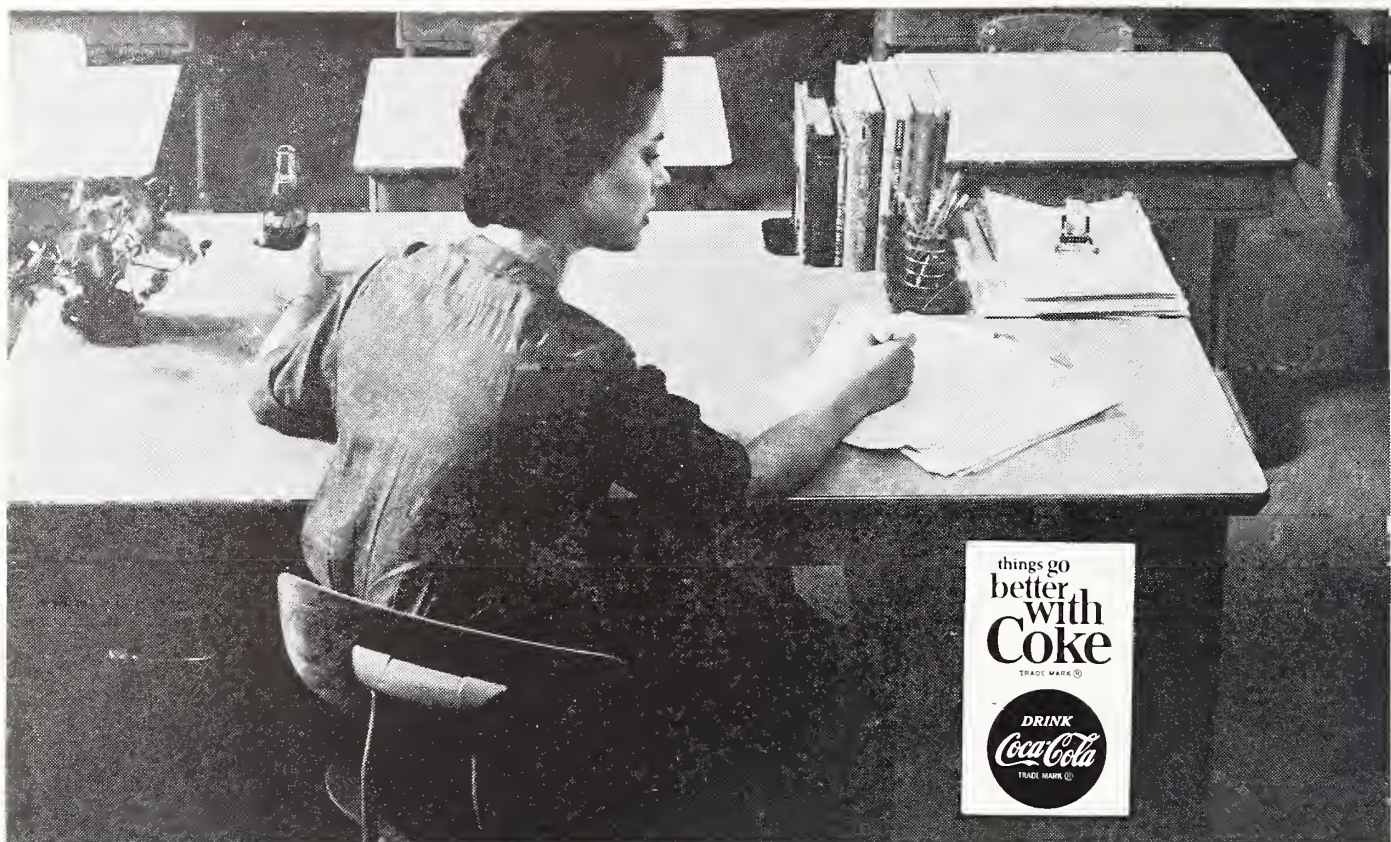
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## The Journal of The Maine Medical Association

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\* AMA Council on Foods and Nutrition: The Regulation of Dietary Fat, *JAMA* 181:411-423 (August 4, 1962).

AMA Council on Foods and Nutrition: Composition of Certain Margarines, *JAMA* 179:719 (March 3, 1962).





# The Journal of the Maine Medical Association

Volume Fifty-Five

Brunswick, Maine, September, 1964

No. 9

## Administration of Parenteral Fluids and Electrolytes to the Surgical Patient

ALFRED HURWITZ, M.D.\*

During the past 20 years many advances in our knowledge of fluid and electrolyte balance have been made. Fluid compartment volume and composition have much greater significance than ever before. The parenteral administration of improper amounts of electrolytes can be attributed to two main factors. First, the doctor may not know the current concepts of parenteral administration because those concepts have been changing rapidly and because many pertinent articles are technical and difficult to comprehend. Second, most of the commercial solutions that were available could not be considered physiologic inasmuch as they did not replace electrolyte losses quantitatively. If the doctor tried to adapt these solutions to appropriate use, he was faced with the onerous task of mixing them in proper proportions. This task was time consuming and impractical.

The purpose of this paper is to review the basic requirements, to discuss the replacement of extraneous losses and finally to describe new aids, e.g. manufactured solutions and a concise "fluid and electrolyte" sheet.

### BASIC REQUIREMENTS

#### 1. WATER

About 60% of the body weight is water. Intracellular water comprises about 40% of the total body weight while the extracellular fluid compartment consists of 20% of the total body weight. Of the latter compartment, only one quarter is intravascular.

During the course of 24 hours, the average adult loses about 1200 ml. of water (750 ml. per square meter

per 24 hours) as insensible water loss and an additional 500 to 800 ml. to excrete nitrogenous waste products through the kidneys. The basic requirements for water in a 70 kg. adult would then be about 2000 ml. daily.

#### 2. SODIUM

In health the average intake of sodium chloride varies from 3 to 15 grams per day depending upon the variation in the use of the salt shaker. The excess sodium is excreted through the kidneys. Since there may be retention of sodium in the postoperative period as a manifestation of adrenal hyperactivity (Selye's "alarm reaction"), one should not administer more than 60 to 80 meq. of sodium per day to satisfy the patient's basic requirement.

If vomiting or diarrhea preceded the patient's admission to the hospital, the preexisting sodium deficiency must be kept in mind. A simple formula that has been of practical benefit is: The amount of sodium to be given in meq. =  $\frac{\text{weight of pt. (in kgms)}}{2} \times (142 - \text{serum sodium level in meq.})$  In the case of a 70 kgm. patient whose serum sodium level is 122 meq. on admission, the amount of sodium to be administered would be  $\frac{70}{2} \times (142 - 122)$  or 700 meq. About one quarter of the requirement could be given during the first 12 hours and the remaining three quarters during the ensuing 2 to 3 days. Too rapid replacement of sodium might throw the patient into marked sodium retention with subsequent peripheral and pulmonary edema.

#### 3. POTASSIUM

Deficiency of potassium should be suspected whenever the intake of food is curtailed. Depletion occurs as a result of urinary and gastrointestinal losses of potassium (vomiting, diarrhea, suction, fistulas). Paradoxically the serum potassium may be normal or abnormally high in

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URINARY FINDINGS

	<i>Specific gravity</i>	<i>Osmolarity milliosmols per liter</i>	<i>Sodium milliequivalent per liter</i>	<i>Blood urea nitrogen</i>
Postoperative water retention	1.015-1.020	600-1000	30-50	normal
Dehydration	1.020-1.030	800-1200	less than 20-30	slightly to moderately elevated
Tubular necrosis	1.010	300	60-80	progressive elevation

dehydration, incomplete rehydration, oliguria and in renal and adrenocortical insufficiency.

The syndrome associated with hypokalemia<sup>1</sup> develops primarily in patients who are being fed parenterally and who have received little or no potassium. Since the basic requirements for potassium in the adult is 40 meq. daily and since the deficit for potassium is a cumulative one, it is important to administer 40 meq. KCl daily to patients who are being fed by the intravenous route only. If the patient has a preexisting deficit because he has not been given potassium then the amount of potassium required to repair the deficit is 40 meq. times the number of days of deprivation. Added to this figure would be the amount of potassium lost extraneously, e. g. in gastric aspirate, biliary or pancreatic losses or intestinal losses. About 10-15 meq. KCl are lost in each liter of the extraneous fluid evacuated. The clinical symptoms of hypokalemia are apathy, lethargy, nervousness, and irritability, muscular weakness, abdominal distention and ileus, occasional confusion, disorientation, delirium, muscular twitching and tetany. The laboratory aids to the diagnosis are flattened T waves and low ST segments on the electrocardiogram and hypokalemia, hypochloremic alkalosis, hypoproteinuria and occasionally hypophosphatemia in the blood chemical determinations. The rate of administration of potassium should not exceed 20 meq. KCl per hour (preferably 5-10 meq. per hour) and probably not more than 250 meq. in any one day in order to avoid the possibility of ventricular fibrillation secondary to potassium toxicity. Because of the known losses of potassium secondary to corticosteroid therapy, an additional 40 - 60 meq. KCl should be given daily to those patients.

4. MAGNESIUM

More recently the significance of hypomagnesemia<sup>2</sup> has come to our attention. The symptoms are progressive weakness, drawing sensations in the fingers and hands, with painful spasm of the muscles of the forearm, and finally tetany. The tetany may be dramatically abolished by the administration of 200 meq. of magnesium chloride intravenously with magnesium supplement by mouth. This type of tetany will not be relieved by the administration of calcium. In fact the serum calcium may be normal while the serum magnesium is low (less than 1.5 meq. per liter).

Hypomagnesemia may be seen in nontropical sprue, after extensive resections of the small bowel, some cases

of cirrhosis, possibly in delirium tremens and in celiac disease.

PRACTICAL CONSIDERATIONS IN THE ADMINISTRATION OF FLUID AND ELECTROLYTES IN THE POSTOPERATIVE PATIENT

Let us assume that an adult patient weighing 70 kg. with no preexisting electrolyte deficits is to undergo a major operation. At the time of operation, blood should be administered in amounts that will replace the estimated blood losses. In this connection, it is well to avoid one-unit transfusions because patients who are not hypovolemic preoperatively can tolerate losses of 500 ml. or less without replacement and the danger of inducing a reaction or serum hepatitis is a real one. Glucose in water are usually administered during the operation and the tubing flushed with saline only (0.33 per cent NaCl solution is ideal) prior to the injection of blood to prevent clumping of the erythrocytes which might occur if blood was added to the glucose solution remaining in the tubing. Postoperatively the patient is given 5% glucose in water in amounts that will not exceed 2000 ml. for the first day. At the end of 24 hours, electrolytes can be safely administered if the patient shows no evidence of tubular necrosis. The above table is helpful in differentiating between postoperative water retention, dehydration and tubular necrosis.

A specific gravity of 1.014 or more together with an adequate urinary output during the first 24 postoperative hours (in excess of 500 ml.) militate against the diagnosis of tubular necrosis. About 50-60 meq. NaCl (300 ml. of 0.9% NaCl and 5% dextrose) should then be administered. Twenty meq. of KCl are added to each of the two liter flasks of 5% dextrose in water. These additive cations will satisfy the patient's basic requirements.

Protein hydrolysates are not given unless the patient must be kept on complete parenteral feedings after the fifth postoperative day or if there has been an uncompensated pre-existing protein deficit. Before the fifth day the patient will not assimilate protein because of the severe catabolic phase in evidence during that period (the adrenergic-corticoid phase-Moore<sup>3</sup>). Chassin<sup>4</sup> has emphasized the fact that when protein hydrolysates are given, the minimal daily dosage should be 150 gms. of the hydrolysate and 300 gm. of carbohydrate. The latter is given to ensure the fact that the protein will not be utilized to replace caloric losses but will be synthesized as



TABLE I

Daily fluid and electrolyte requirements are the sum of the Basic Needs and the Replacement of Extraneous Losses

Basic Needs cover urine, sweat and insensible water loss. Estimate 35 Cal/Kg plus 7% for each degree of fever. Requirements per 100 Cal are: Water 90 cc-100cc Na, Cl 1-2 mEq K 2-3 mEq		Replacement of Extraneous Losses must be in kind as measured. Average content in mEq/Liter is as follows:  Gastric Asp 80 12 150 0 Intest Fluid 140 10 105 25 Bile 140 10 100 30 Pancreatic Juice 140 10 75 75		Electrolyte content of commonly used Solutions — mEq/Liter  Solutions NA Cl Lact HCO Isotonic Saline (0.9%) 154 154 0 0 Hypotonic Saline 77 77 0 0 Hypertonic Sal. (5.0%) 850 850 0 0 1/6 M Lactate 166 0 166 0 Sodium Bicarbonate (1.5%) 178 0 0 178			
Parenteral Vitamin Supplementation should be given daily and satisfy the requirements below: Ascorbic acid 300 mg Thiamin HCl 5 mg Riboflavin 5 mg Niacinamide 100 mg Calcium pantothenate 20 mg Pyridoxine 2 mg Folic acid 1.5 mg Vit B <sub>12</sub> 1 mg  Caloric Calculations Substance provides Cal per Gm Carbohydrate 4 Protein (amino acids) 4 Alcohol 7		Abbott Solutions G and D should be used for quantitative replacement of gastrointestinal fluid losses. Soln G replaces gastric secretions. Soln D replaces intestinal fluid, bile and pancreatic secretions. These solutions provide in mEq/L  NaCl KC1 NH <sub>4</sub> CL NaLactate Soln G 63 17 70 0 Soln D 88 12 0 50 Conversions Aids 1 Gm NaCl 17 mEq of Na & Cl 1 Gm KC1 13 mEq of K & Cl 1 Mm NH <sub>4</sub> Cl 18.7 mEq of NH <sub>4</sub> & Cl		Speed of Infusion and Rate of Utilization  1) No more than 20 mEq of K per liter of solution per hour should be given IV 2) Substance Rate of Utilization 5% Glucose 750 cc per 1 hour 10% Glucose 375 cc per 1 hour 10% Invert Sugar 900 cc per 1 hour 5% Alcohol 1000 cc per 3 hours			
				Electrolyte Composition of Blood Plasma  (Cations) + (Anions) — Base Ions, mEq/L Acid Ions, mEq/L Na 142 HOC <sub>2</sub> 27 K 5 Cl 103 Ca 5 HPO <sub>4</sub> 2 Mg 3 SO <sub>4</sub> 1 155 Organic acids 6 Protein 16 155			

- 1) As a rule replacement of basic K needs should not be attempted during the first 24 hours post-op.  
K replacement is started thereafter only when urine output is adequate.
- 2) Na Basic needs should be under-replaced during the first few post-op days because of renal retention of Na.
- 3) If the period of intestinal fluid loss is prolonged or the losses are extraordinarily large, parenteral Ca gluconate should be given 1 Gm per day.
- 4) The following formula is used to calculate replacement of sodium in deficits that exist before therapy or develop in spite of it: Amount of ion =  $\frac{\text{pt's wt in Kg}}{2} \times \frac{(\text{normal serum value} - \text{pt's serum value})}{(\text{of ion in mEq})}$  (required in mEq)

tissue protein and lean muscle. Elman<sup>5</sup> was able to maintain patients in positive nitrogen balance on parenteral feedings for many weeks by adhering to the above-mentioned doses. The administration of vitamins to postoperative patients is usually unnecessary unless the patient showed evidence of nutritional deficiencies prior to operation or if he required prolonged parenteral feedings postoperatively. Vitamin B and C should be administered because they are the vitamins most essential for the surgical patient. As a means of assuring adequate replacement therapy, a chart has been devised (Table 1). The chart stresses the general concept that the fluid and electrolyte requirements for any patient depend on his basic needs and extraneous losses. The formulae for basic requirements are listed in the upper left hand corner of the chart. Adjustments are made for the weight of the patient and for the degree of hyperpyrexia. The amounts of electrolytes lost via extraneous routes are depicted in the middle column. It will be noted that the requirements for replacement of electrolytes lost by gastric aspiration differ from those lost through bile, pancreatic and intestinal fistulae in that the former is

characterized by large losses of chloride (far in excess of the sodium losses), whereas the latter group lose large amounts of sodium and other base. Two types of solution have been devised, one of which aims at replacement of losses secondary to gastric aspiration which would be especially pronounced in the patient with an obstructing duodenal ulcer. The other solution was designed to replace losses from the small intestine, e.g., those due to ileostomy, pancreatic or bile fistulae or drainage from a Miller-Abbott tube placed in the small intestine. Experimental and clinical observations reveal that for the most part, replacement of all electrolyte and fluid losses can be accomplished by replenishing the basic needs of the patient on the one hand, and adding quantitatively solution G or solution D\* to replace the measured extraneous losses on the other hand. This chart has proven most useful, not only as a guide to treatment, but also as an instructional aid to the physician.<sup>6</sup> It is maintained on a day-by-day basis (Table

\*These solutions are manufactured by several companies to conform to the composition depicted on Table 1.

TABLE II

Date	Temp Elev of	Wt in Kg	In Out	P	BASIC REQUIREMENTS					Extraneous Losses		Urine Out- put	Fluids Given		Lab. Data & Remarks etc		Name	
					Calories CHO	TOT	Water CC	Na	Cl	K	Type		Amt.	Type				Amt
			In										B		NPN CO <sub>2</sub> Cl Na K	HMC Hb Prot	Bal. Sheet No.	
			Bal															
			Out										B		NPN CO <sub>2</sub> Cl Na K	HMC Hb Prot		
			In										X				Md.	
			Bal															
			Out										B		NPN CO <sub>2</sub> Cl Na K	HMC Hb Prot		
			In										X				Ref. No.	
			Bal															
			Out										B		NPN CO <sub>2</sub> Cl Na K	HMC Hb Prot		
			In										X				Name	
			Bal															
			Out										B		NPN CO <sub>2</sub> Cl Na K	HMC Hb Prot		
			In										X					
			Bal															

"Out" refers to amounts required to meet basic needs as determined from Table I. The average adult will require about 2000 ml. of fluid, 60 meq. Na and 40 meq. K. In the "In" column the exact amounts of each electrolyte that the patient received that day are recorded.

II) and should help prevent a cumulative deficit or an excess of any of the constituents. Since it is a permanent part of the patient's record, it is available for a comprehensive study of parenteral alimentation at any time.

PRACTICAL POINTS IN THE ADMINISTRATION OF FLUIDS AND ELECTROLYTES

1. Order the solutions to be started at 8 or 9 a.m. by inscribing the order during the previous evening.
2. Number all solutions to be given in any one day serially: 1, 2, 3, etc.
3. In addition to the solutions given as basic requirements (usually two liters) administer the appropriate solutions to replace extraneous losses in a quantitative manner.
4. The rate of administration should be approximately four hours per liter. This rate will ensure proper utilization of carbohydrates and amino acids and will prevent the possibility of potassium toxicity even if 40 meq. KCl are added to a liter of the solution. If the patient has cardiac disease, the rate should be much slower (8 to 10 hours per liter).
5. In most instances the required solutions can be injected in 12 to 15 hours (completed by 10 or 11 P.M.) and the needle withdrawn. In this way the same vein will be available for infusion on the following morning. If a cutdown was necessary, patency of the polyethylene tubing can be ensured by injecting 10 mg. of heparin into the tubing at the culmination of the daily infusion.

6. If parenteral alimentation is necessary for a protracted period, employ 3000 ml. of 5% protein hydrolysate and 10% glucose to satisfy the patient's basic requirements.
7. Break the seal to insert additional solutions just prior to the administration of that bottle to minimize proliferation of bacteria that might have been injected inadvertently.
8. Add vitamins only if a preexisting deficit exists or if prolonged parenteral feedings become necessary.
9. Employ magnesium in the treatment of patients who are candidates for the development of hypomagnesemia, e.g. non-tropical sprue, extensive resections of the small bowel and in the diarrheal states.
10. A urine specific gravity of 1.014 or more and urine urea nitrogen: serum urea nitrogen ratio of more than 10 usually denote satisfactory renal function.

CONCLUSIONS

1. A modus operandi for the replacement of fluids and electrolytes emphasizing the basic requirements on the one hand and the extraneous losses on the other has been described.
2. Newer types of solutions have been devised to combat the latter deficits.
3. The administration of protein hydrolysates and of magnesium may become mandatory in depleted patients.

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# Carcinoma of the Female Breast

## Associated with Pregnancy and Lactation

EUGENE P. McMANAMY, M.D.\*

### INTRODUCTION

Carcinoma in the female breast during pregnancy or during the lactation period is a serious complication of a normal physiological state, and as such should be promptly and adequately treated in an orthodox manner. One has only to glance through the literature written over the past thirty years to appreciate the changes in the management of this still somewhat controversial subject. In recent years definite ground rules based on rational foundations are gradually being formulated. Predominate is the rule of treating the case of cancer of the breast associated with pregnancy or lactation the same way as when there is no association of these conditions with cancer of the breast.

The somber outlook of thirty years ago is well pictured in an excellent monograph prepared by Dr. Grantly Taylor,<sup>1</sup> at the request of the committee on the treatment of malignant diseases, American College of Surgeons.

Taylor<sup>1</sup> quoted Sistrunk, McCarty, and Harrington as stating that carcinoma which developed during pregnancy or during the lactation period invariably proved fatal within five years after operation, and that operation is not justifiable in such cases, as the prognosis is hopeless.

In 1943, Haagensen and Stout<sup>2,3</sup> placed pregnancy associated with breast cancer first among the nine points of inoperability. (Unfortunately, their conclusions were based upon the results of their management of only five cases.)

In 1949, Barney Brooks and Proffitt<sup>4</sup> interjected some hope into the controversial field by concluding that malignant lesions of the breast co-existing with pregnancy and lactation were operable, but that such cases carried a less favorable prognosis than in non-pregnant women with breast cancer.

In 1952, after reviewing 136 cases, Harrington<sup>5</sup> assumed a much more optimistic attitude towards the management of cases of breast cancer when it coincided with pregnancy or lactation.

Shortly thereafter, Adair,<sup>6</sup> and Westberg<sup>7</sup> reported their figures on the survival rate of patients treated for this complicated problem.

In 1955, White<sup>8</sup> published the results of treatment of his own cases as well as those from the world literature.

The survival rates of treated breast cancer as reported by Harrington,<sup>5</sup> Abair,<sup>6</sup> Westberg,<sup>7</sup> White,<sup>8</sup> and Byrd<sup>9</sup>

TABLE I

Author	Analysis of Collected Series							
	NON-PREGNANT				PREG. & LACT.			
	Breast		Brst. & Ax.		Breast		Brst. & Ax.	
	5	10	5	10	5	10	5	10
Harrington <sup>5</sup>	78.3	63	32.5	15	64.5	54.2	8.9	5.4
Adair <sup>6</sup>	77.5	54.5	39.4	21.2	71	—	27.4	—
Westberg <sup>7</sup>					63.3	55.2	—	7.3
White <sup>8</sup>					72.8	26.3	6.3	0
Byrd <sup>9</sup>					100	—	28	—
Average	77.9	58.7	35.9	18.1	74.3	45.2	17.6	6.3

as given in Table I serve as a good comparison between non-pregnant and pregnant groups, and tend to reflect a more optimistic attitude than previously held.

In studying the average percentages as expressed in the bottom line of Table 1, one is favorably impressed by the fact that the figures in the pregnant and non-pregnant groups are comparable in both the 5 and 10-year column when the disease is confined to the breast alone. When the breast and axilla are both involved at the time of definitive treatment, the survival rates for the pregnant and lactating group leave much to be desired.

In the light of present knowledge, it is most difficult to hold the physiological state of pregnancy solely responsible for the poor survival rate in this particular group of cases.

Other factors must be sought in dealing with this problem.

### FACTORS AFFECTING THE PROGNOSIS

Many factors have been advanced as affecting the prognosis in these cases of pregnancy and lactation complicated by breast cancer. Some of these factors are presented as follows:

#### 1. Advanced Stage Of The Disease

The advanced stage of the breast cancer in pregnant and lactating women at the time of definitive treatment can be appreciated by reviewing the series of the various authors that was presented in Table 1. Metastatic disease was found in 298 of 424 cases or in 70% of the 5- and 10-year survivals.

#### 2. Delay In Diagnosis And Treatment

Always a distressing fact is the common finding of a lag period between discovery of a lump in the breast and treatment of it in these cases.

Delays have been reported as varying from 2 to 8 months, and in some instances to several years. The responsibility must be shared between patient and physician.

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Westberg<sup>7</sup> studied parallel groups of women with breast cancer complicated and uncomplicated by pregnancy. The average woman reported her tumor to the physician two months later if she was pregnant or nursing than if she were not. After recognizing the mass, the doctor delayed an average of a month longer in the treatment of a patient who was pregnant or nursing than in one without these complications.

Bunker and Peters<sup>10</sup> found a definite lag period in diagnosis in their study of 150 cases with a disproportionately long interval between discovery of a lump and the actual diagnosis. Only 10 out of 150 cases were diagnosed as having breast cancer within 1 month after discovery of the lump in the breast. Less than one-third of the patients had the diagnosis confirmed within three months. Three cases were allowed to go 5 years before the diagnosis was made.

Montgomery<sup>11</sup> has reported that in his series the pregnant patient was first to find the lump in her breast 90% of the time. This is at a time when the patient is making regular visits to the physician's office for routine prenatal checkups.

A personal survey was recently made of most of the physicians who practice obstetrics in a community to determine how frequently pregnant patients received breast examinations during their routine prenatal and postnatal visits. All stated that they performed breast examination on the first prenatal visit and not again until the sixth or eighth week post partum checkup.

The delay in diagnosis apparently arises from three sources; namely, failure to realize that breast cancer occurs during pregnancy, failure to examine the breasts regularly, and failure to promptly biopsy any lesion that is found.

### 3. *Masking Of The Tumor*

The rapidly expanding and engorging breast during pregnancy and lactation tends to obscure nodularity, making it difficult to differentiate physiological hypertrophy and pathological nodularity.

### 4. *The Youth Of The Patient*

The average age of the pregnant patient who has mammary cancer is 34 years. This particular fact may have more bearing on the prognosis in such a case than is commonly appreciated.

Haagensen<sup>12</sup> and his associates, in their recent report of a personal series of 556 patients with breast cancer, found a poorer prognosis and an increased malignancy in premenopausal women, particularly in the younger age group, when compared with postmenopausal women. There was a 66% incidence of axillary metastases in those patients under 36 years of age treated for breast malignancy, and only a 48% incidence in the postmenopausal group. The 5-year survival rates in the clinical stage A group (those with disease only in the breast tissue itself) was 92% in the postmenopausal, 82% in the premenopausal, and only 53% in those under 36 years of age.

These figures would tend to place women under 36

years of age with breast cancer in a group with a relatively poor prognosis whether or not pregnancy or lactation co-existed.

### 5. *Hormonal Effect*

According to Beck and Rosenthal,<sup>13</sup> pregnancy greatly accelerates the development of the duct system, and at the time of lactation the capillaries and lymphatics of the breast are engorged.

Estrogen is a specific growth stimulant to the normal mammary epithelium, and theoretically might be expected to promote the growth of cancer during pregnancy. But, as stated by Holleb and Farrow<sup>14</sup> and Austin,<sup>15</sup> a great deal has been surmised, but proof is lacking that there is an increase of mitotic activity in human beings or that mammary cancer may become more anaplastic during the proliferative phase. The clinical effect of circulating estrogens on cancer in the lactating breast cannot always be demonstrated, according to Peters,<sup>16</sup> who has found contradictions in the course of such complicated cases.

## INCIDENCE

There is an incidence of carcinoma of the breast of three per 10,000 pregnancies, and 2.8% of the cases of breast cancer are associated with pregnancy.

### REVIEW OF CASES — DATA ON 8

A review of the medical records of two local hospitals from 1951 to 1961 yielded 8 cases in which pregnancy or lactation was associated with primary breast cancer or with recurrent malignancy.

The average age of patients studied was 32 years; the youngest was 25; and the oldest was 39 years.

There was an average delay of 8 months between the time that the lesion in the breast was first found until definitive treatment was instituted.

Four cases received radical mastectomy during pregnancy and one during the lactating period. One patient died 2 3/4 years after mastectomy; the other 4 have survived 2 1/2, 3, 11, and 14 years after mastectomy. There were positive nodes in 3 of the 5 mastectomy cases.

One patient was classed as having inoperable breast cancer at the time of pregnancy. Two cases had had mastectomies previously and subsequently developed recurrences and pregnancies. These 3 were treated by pregnancy interruptions and castration. All 3 are dead, having survived 4 1/2, 1 1/2, and 1 year respectively after castration.

Bilateral breast involvement was found in 3 cases, metastatic in 2, and a new primary in the third.

One patient has been delivered of 2 normal pregnancies subsequent to mastectomy and is alive and well after 14 years.

CASE 1. Aged 38. 8 children. Lump left breast for 11 months, which grew faster the last two months. Erythema, heat and tenderness were found over the mass. The patient was 6 1/2 months pregnant. 7/14/58 radical mastectomy was performed. Carcinoma Simplex with 20 negative nodes. 10/5/58 delivery of a normal child 3/17/60 recurrent disease. 3/19/61 death due to metastatic disease. This patient lived 2 3/4 years after mastectomy. She perhaps could be classed as having very early inflammatory disease. The chief problem here was the delay on the part of the patient in seeking help for her breast cancer.

CASE 2. Aged 29. 3 children. 11/28/61 lump in her left breast for 1 week. Patient was 4 months pregnant. 12/28/61 radical mastectomy was performed. Adenocarcinoma with nega-



tive nodes. 5/26/62 normal delivery. This patient is alive and well and there is no evidence of recurrence of disease.

CASE 3. Aged 39. 2 children. Lump in the right breast for 10 months; now fungating mass with axillary and supraclavicular metastases. Patient was 6 1/2 months pregnant. 3/17/61 simple mastectomy was performed. May and June, 1961, patient was given a course of x-ray therapy (2,000 R). 6/27/61 mass left breast noted at time of hospital admission, which was not present at the time of mastectomy in March. 6/28/61 normal delivery. 6/30/61 biopsy excision, left breast revealed adenocystic mastopathy, metastatic carcinoma. Bilateral oophorectomy was done. This patient is alive and well, and no evidence of recurrence has been found.

CASE 4. Aged 25. 2 children. 12/1/52 mass in left breast. 3/8/53 patient was 2 1/2 months pregnant. 3/9/53 radical mastectomy. Intraductal carcinoma of the left breast of high grade malignancy. 17 negative axillary nodes. 9/13/53 normal delivery. Seven years later (2/15/60), tumor, right breast was found. 2/24/60 right radical mastectomy was performed. Adenocarcinoma with 10 negative nodes. The patient is alive and well and there is no evidence of recurrence of cancer.

These four cases are the only ones that actually had mastectomy at the time of pregnancy. Case 1 survived only 2 3/4 years after mastectomy. Cases 2 and 3 are free of disease 2 1/2 and 3 years respectively, but are too recent in history to warrant comment, except perhaps for Case 3, who has shown strong host resistance to cancer so far, having had a simple mastectomy with x-ray therapy to the right side and biopsy excision on the left side with oophorectomy and no further x-ray therapy since, and no evidence of disease at present. Case 4 is living and well, free of disease 11 years after mastectomy, left side, and 4 years after mastectomy for a new primary on the right side. This patient is a good example of proper management and treatment and possibly strong host resistance to malignancy.

CASE 5. Aged 36. 5 children. 9/15/50 2 weeks postpartum patient noticed a lump in her left breast. 11/8/50 left radical mastectomy was performed. Intraductal carcinoma of the left breast showing lymphatic invasion and axillary node metastases. 1955 pregnancy with normal delivery. 1961 pregnancy with normal delivery. The patient is still alive and well with no evidence of recurrence, 13 1/2 years after radical mastectomy.

CASE 6. Aged 29. 5 children. Mass in left breast for two months. 12/9/51 radical mastectomy was performed. Medullary carcinoma with positive axillary nodes. 6/52 recurrence in the scar which was excised and later treated by x-ray therapy with complete regression. 10/52 a four-week pregnancy was interrupted and a castration was performed. In 1954 there was a recurrence in the parasternal nodes. In 1955 a tumor of the right breast with metastatic disease in the right axilla and supraclavicular area was found. The patient was treated by x-ray therapy. The patient expired in 1957 due to carcinomatosis. This patient lived 6 years after radical mastectomy. 4 1/2 years after abortion and castration.

CASE 7. Aged 35. 2 children. 3/10/53 patient noticed a lump in the left breast shortly after being struck in the breast. 6/30/53 left radical mastectomy was performed. Scirrhus carcihoma, grade III with one positive axillary lymph node. 11/28/53 metastatic supraclavicular node of 2 weeks duration was noted. Patient was 4 months pregnant. 11/30/53 therapeutic abortion and oophorectomy was done. 9/30/54 onset of pleuritic pain with evidence of pulmonary metastases. 11/7/54 the patient expired from carcinomatosis. This patient survived 1 1/2 years after radical mastectomy and approximately 1 year after interruption of pregnancy and oophorectomy.

Cases 5, 6, and 7 are examples of pregnancy developing after completion of radical mastectomy.

Two of the cases are good examples of rear-guard action in which the maternal salvage hardly justified the fetal wastage.

It has been estimated that 1 patient in 19 of those in the child bearing period will become pregnant subsequent to

TABLE 2

Survival Rate in Pregnancy after Mastectomy SURVIVAL IN PREGNANCY AFTER MASTECTOMY				
Author	Without metastases		With metastases	
	5 years	10 years	5 years	10 years
White <sup>8</sup> 268	64.6	51	43.8	34.6
Bunker <sup>10</sup> 25	90.9	—	70	—
Brown <sup>17</sup> 20	81.2	56.2	50	25
Average non-preg. Table 1.	77.9	58.7	35.9	18.1

radical mastectomy. This probably accounts for the lack of any large reportable series.

The survival rates recently reported in the literature as shown in Table 2, reveal that the prognosis is quite favorable in this particular group of cases.

The figures at the bottom of the table represent those of the average percentage of the 5- and 10-year survival rate in the non-pregnant group as shown in Table 1. After comparing these figures, one may readily assume that the prognosis in mastectomy treated cases is not adversely influenced by subsequent pregnancy. On the contrary, it would appear that these cases are enhanced.

Controversy is still in evidence over the management of this group of patients. Statistically, there is no valid evidence that abortion will improve the survival rate or change the course of the disease. Yet the adherents of therapeutic abortion steadfastly maintain their stand on purely theoretical grounds even in the face of mounting evidence against the benefit of such procedures.

CASE 8. Aged 34. This patient had been married for 10 years and never before pregnant. 1/29/61 the patient gave a history of having had a lump in her left breast for 2 years and a pregnancy of 4 months duration. Physical examination was negative except for peau d'orange and edema of the skin of the left breast overlying an 11 cm mass. This mass was moveable on the chest wall. There were palpable nodes fixed high in the left axilla. Pelvic examination revealed an enlarged uterus consistent with a 4 1/2 month pregnancy. Osseous metastases to ribs and to L. 4&5 were visible by x-ray study. 1/31/61 needle aspiration biopsy of the left breast mass revealed the presence of a poorly differentiated carcinoma.

Radical mastectomy was considered contraindicated, and on 2/1/61 an abdominal hysterectomy and oophorectomy was performed as palliation. The uterus was found to contain a 4 1/2 to 5 month pregnancy.

Postoperatively the patient received x-ray therapy to skeletal metastases. 10/30/61 the metastatic disease expanded and now included the liver as well as other skeletal centers of involvement. Bilateral adrenalectomy was performed at this time. The patient continued on her down-hill course and expired on 5/29/62 from carcinomatosis. This patient had survived 1 year and 4 months following abortion and castration.

This case was obviously hopeless from the start. The situation was desperate and the management was futile.

Hysterectomy and oophorectomy seemed rather harsh treatment and hardly justifiable, for the operation caused interruption of the pregnancy, assuring an eventual 100% mortality instead of 50%, and it also denied the joys of parenthood to a childless couple married ten years.

It could be argued that the morbidity from normal delivery or early Cesarean section would hardly be greater than the morbidity from the two major surgical procedures to which she was subjected in the remaining days of her life.

In retrospect, in the words of Barney Brooks,<sup>4</sup> one can well

imagine that the sum total of happiness of a childless woman living 16 months after interruption of her pregnancy, and subsequent adrenalectomy, might be a whole lot less than her living 16 months, if this were composed of the 5 remaining months of anticipation, and 11 months of realization of having accomplished her most primitive obligation for the survival of the human race.

CONCLUSIONS

1. There seems to be agreement generally over the management of cases of breast cancer complicating pregnancy or lactation.
2. Controversy exists over the proper care of some of the more complicated cases and where treatment of breast carcinoma has been completed prior to pregnancy.
3. Frequent, careful breast examination of women in the pre-and postnatal checkups is essential.
4. Early diagnosis and effective, prompt therapy of all breast tumors associated with pregnancy and lactation should achieve equivalent results to those in the non-complicated patients of the same age.
5. Pregnancies subsequent to completed treatment of breast cancer do not adversely influence the prognosis.
6. The average age of the pregnant patient who has associated cancer of the breast is 34 years.
7. Reported statistics would tend to place women under 36 years of age who have breast carcinoma into

a group with a relatively poor prognosis whether or not pregnancy or lactation co-existed.

8. There is no statistical evidence of the therapeutic value of therapeutic abortion.

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ADMINISTRATION OF PARENTERAL FLUIDS AND ELECTROLYTES TO THE SURGICAL PATIENT —  
*Continued from Page 162*

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# Surgical Treatment of Massive Pulmonary Embolization — Report of a Case

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JAMES H. BONNEY, M.D., and RICHARD C. DILLIHUNT, M.D.\*

The diagnosis of acute massive pulmonary embolism must be strongly considered when the classical signs of dyspnea, cyanosis, and tachycardia occur suddenly. When symptoms including chest pain and apprehension — frequently an expression of profound fear of impending disaster — are also manifest, the diagnosis becomes even more likely. Other signs which may or may not appear include progressive distention of cervical veins, hemoptysis and hypotension. Though often not present, evidence of a possible source of emboli such as leg swelling and tenderness may point toward the diagnosis of embolism. Frequently the patient presenting the above picture has recently experienced accidental trauma or undergone surgery.

When time permits, x-ray examination may be helpful in establishing the diagnosis. The routine chest x-ray may show relative radiolucency or decreased vascularity of peripheral lung fields. Some investigators<sup>1</sup> have advocated clinical application of pulmonary arteriography in reaching a definitive diagnosis. This may be helpful in the patient whose diagnosis is less certain, and who stabilizes sufficiently to permit this procedure. Electrocardiographic evidence of cor pulmonale has been helpful in diagnosis — especially if serial tracings are available. A normal or only slightly elevated SGOT along with LDH and bilirubin elevations have been shown to help differentiate this condition from acute myocardial infarction and pneumonia — the two conditions most commonly considered in the differential diagnosis.<sup>2</sup> Unfortunately all these studies can rarely be performed when massive embolism has occurred, as the patient is usually moribund, and time does not permit. A rapid clinical evaluation of the patient must often dictate the steps which one should follow in establishing a diagnosis and instituting treatment.

The treatment of massive pulmonary embolism may be divided into medical and surgical categories. The medical management is not standardized, but certain approaches appear to be beneficial. Vasopressors are often used, but may in fact be contraindicated as suggested by de Takats.<sup>3</sup> Medical measures have been much more rewarding in treatment of lesser emboli and in prophylaxis against embolism than in the treatment of massive embolism. Long term anticoagulation has been

clearly shown to be beneficial in the embolus prone individual.<sup>4,5</sup>

The surgical treatment of acute massive pulmonary embolism by pulmonary embolectomy was first proposed by Trendelenberg in 1908,<sup>6</sup> and the first successful case was reportedly performed by Kirschner in 1924.<sup>7</sup> A small number of successful embolectomies were accomplished in Europe during the 1920's and 1930's. With the advent of anticoagulation, the enthusiasm for surgical treatment was dampened until the report of Steenburg et al of a successful embolectomy at the Peter Bent Brigham Hospital in 1958.<sup>7</sup> This apparently was the first successful embolectomy in the United States, and was responsible for a renewed interest in the procedure. The use of cardiopulmonary bypass during embolectomy was introduced by Sharp et al<sup>8</sup> in 1961, and following this original effort other groups reported success using this refinement.<sup>9,10,11,12,13</sup> The fact that at least seven successful embolectomies utilizing extracorporeal circulation have been reported in recent years is convincing evidence that embolectomy has been established as the treatment of choice in certain selected cases. That this procedure is entirely feasible is again illustrated by our case:

On August 28, 1963, a 60-year-old white female underwent uneventful cholecystectomy for chronic cholecystitis and cholelithiasis at the Maine Medical Center. Her prior history was non-pertinent except for a bout of phlebitis in the left leg some 22 years prior to admission. Postoperatively she ambulated on the 3rd day and progressed satisfactorily until the 10th day when she suddenly felt weak and collapsed on her bed upon returning from the bathroom. She complained of nausea and a tight feeling in her chest. Examination at this time revealed P 100, R 28, BP 90/60, she appeared acutely ill and was short of breath. Heart and lungs were normal, and there was a positive Homan sign on the left, plus calf and femoral triangle tenderness.

Shortly thereafter she became profoundly hypotensive, neck veins were distended, and cyanosis appeared. EKG (as compared to previous EKG) showed sinus tachycardia, appearance of prominent S waves in leads I and AVL, as well as Q waves in lead III (Fig. 1). This pattern was felt to be highly suggestive of pulmonary embolization.

Oxygen and pressors were given and the patient was digitalized, despite this she continued in profound shock (blood pressure unobtainable much of the time). Chest x-ray was negative, CBC revealed Hb 12.2, Hct 39, WBC 14,900 with a moderate left shift. Transaminase was 400 units, lactic dehydrogenase 840 units, bilirubin 4.4 mgm.%.

On September 8, 1963, twenty-five hours after the onset of symptoms, the patient was anesthetized and hypothermia commenced, bringing the temperature to 90°F. Cardio-pulmonary bypass was then instituted via the right femoral artery and

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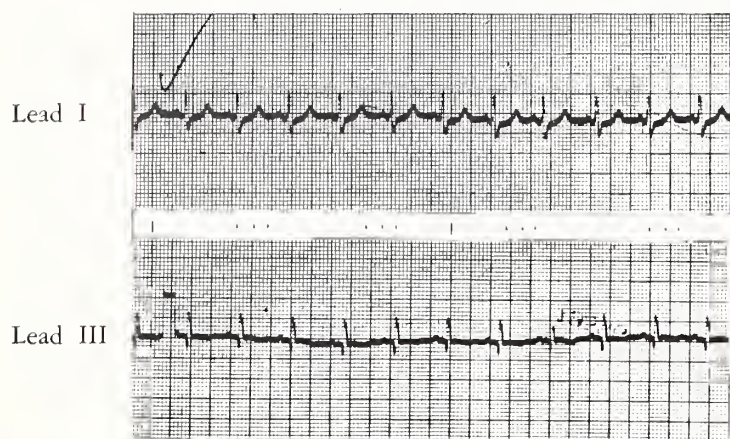
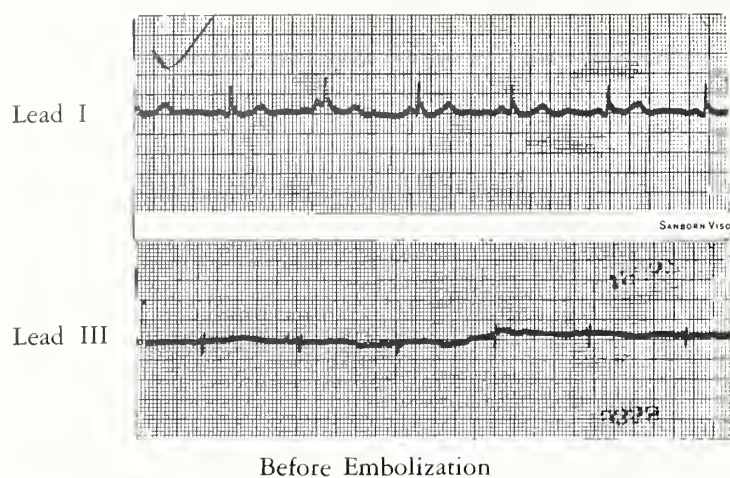


FIG. 1

vein utilizing pediatric disposable bubble oxygenator with flow rate of 900 cc/min. The chest was then entered through a transverse sternal transecting incision at the third interspace. The right heart was tense and distended, the pulmonary artery grossly dilated. Inflow was shunted to the oxygenator, the artery opened, and multiple clots removed from its branches (Fig. 2). Transient arrhythmia occurred, followed by conversion to normal rhythm. Blood pressure rose to normal and the procedure was concluded with bilateral femoral vein ligation.

Anticoagulation, instituted preoperatively, was continued postoperatively. She had a benign postoperative course, ambulating on the 13th day and was discharged home on the 20th day — asymptomatic except for mild generalized weakness. The patient had an uneventful convalescence, and when last seen seven months post embolectomy, had resumed housework and complained only of mild intermittent swelling of feet and ankles.

Thus we add this case to the small but growing list of successful embolectomies. With new developments and improvements in anesthesia, blood replacement, hypothermia, and extracorporeal circulation, it has become evident that embolectomy now occupies a much stronger position in the treatment of this catastrophic occurrence. It is our feeling that cardiopulmonary bypass should be utilized if available, but previous workers have shown that this technique is not absolutely necessary.

We feel that when a patient experiences a catastrophe, which on initial evaluation appears to be a massive embolus, there is little or no time to delay. Laboratory data and other diagnostic aids must be obtained immediately. The operating room must be alerted as soon as em-

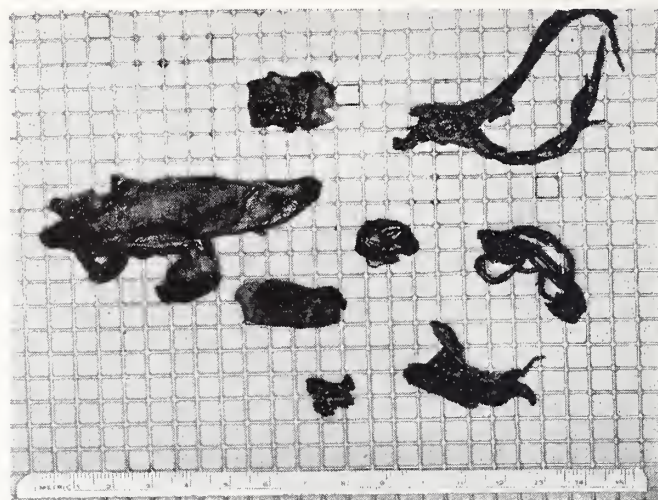


FIG. 2

bolectomy is considered. The patient must be made ready for immediate surgery. Close observation will then dictate management. Some will be found to have had some other occurrence. Others will be found to have had a lesser embolus which responds well to conservative management. Of those who have had massive emboli, many will die within minutes, but a small group will survive long enough for the diagnosis to be established and to be transferred to surgery. If surgery is not performed, a few may survive, but they can be expected to go on to severe chronic pulmonary incapacitation in the future.

Once the diagnosis is established beyond reasonable doubt, and it is clinically evident that the patient is deteriorating — and cannot survive with conservative measures — the time has arrived for immediate thoracotomy and embolectomy. We feel that a small number of patients can be saved from imminent death when managed in this fashion.

#### SUMMARY

This report presents a brief review of the syndrome of massive pulmonary embolization. A discussion of the diagnostic steps which may be helpful is followed by a case report of a successful embolectomy. A basic plan of action is described whereby the patient who has experienced a massive pulmonary embolus becomes a candidate for surgery.

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# Enzymatic Debridement of Third Degree Burns in Animals with Bromelains—A Preliminary Report

GEROLD K. V. KLEIN, M.D.

With all the progress in therapeutics, the fate of severe third degree burn victims has only little improved. The realization of this fact becomes of great concern when considering any possible future mass catastrophe, in which a large number of casualties will be burn victims.

One of the major reasons for the slow change in the prognosis of major burns is the fact that, in spite of all the developments in the field of infection control, we are still unable to prevent septicemia which, in the majority of the severely burned patients, is the usual cause of death. Since the devitalized tissue of the eschar — an excellent culture medium — is the main source of septicemia, it is generally agreed that prevention of infection by early debridement would be a good therapeutic measure.<sup>4,17,23,24,36,39</sup>

It is beyond the scope of this preliminary report to describe all the attempts previously made to accomplish early debridement. Briefly, these attempts can be classified as mechanical, chemical and biochemical. The numerous chemical attempts such as tannic acid, salicylic acid, pyruvic acid, etc., just to mention a few, were discarded since they all in some way proved to be injurious to the already damaged tissue.<sup>18,23,24,36</sup> Intensive investigation has been carried on with biochemical agents to achieve enzymatic debridement. Papain, Pinguinain, Ficin, Trypsin, Streptokinase, Fibrinolysin and other enzymes have been and are still under thorough investigation.<sup>2,11,13,14,15,16,19,20,21,25,26,27,31,34,37,38,40,41,42,43,44,45</sup> However, the general opinion has been expressed by Artz, as follows: "None of the enzymes used today seem to have any practical value. They are quite expensive and seldom remove the eschar any quicker than saline soaks."<sup>4</sup>

With this almost generally accepted opinion the emphasis in early debridement has depended on hydration in various forms: hubbard tanks, wet dressings, etc.<sup>4,30,36,39</sup> None of these methods remove the eschar early enough to prevent infection. Only the sharp, surgical debridement, lately widely practiced, has been able to accomplish this.<sup>3,4,8,29</sup> It is extremely valuable in the treatment of small deep third degree burn surfaces, provided it can be exercised by a highly skilled team, able to judge the depth of the burn, and provided there is sufficient blood available to replace the usually extensive blood loss. These criteria do not exist in mass catastrophes. Furthermore, this procedure can only be applied to small surfaces, since it is extremely shocking to the already shocked patient. Finally, it does require full anesthesia, with all its hazards, for a severely burned patient in his delicate condition.

The author had attempted, in 1956 at the Childrens Hospital, Washington, D. C., and the Washington Hospital Center, to improve the mechanical debridement by hydration through an intermittent shower with normal saline, with the undressed patient placed in a closed plastic tent.<sup>30</sup> This method seemed to speed the process of maceration and slow down the progress of infection. However, the time gained by this technique was not sufficient to eliminate infection completely. Consequently, the author suggested the addition of enzymes to this hydration approach. Unfortunately the enzymes then available seemed to be expensive and, therefore, unfeasible for general use. In addition, the published investigational work seemed to indicate that there was not much time gain over the hydration debridement alone.<sup>36</sup> Some of the enzymes also seemed to have toxic side-effects and attack viable tissue.<sup>31</sup>

In spite of these discouraging reports, further search for a useable enzyme seemed indicated. Its properties had to satisfy the following: (1) Debridement of a third degree burn in less than two days; (2) Proteolytic activity limited to devitalized tissue only; (3) Non-toxicity; (4) Simplicity in application. In addition to these prerequisites, it was desirable that this agent be as painless as possible. (However, for a short period, a patient could be kept heavily sedated without risking the hazards of full anesthesia.) It should be easily stored, and low in cost.

At least three of these qualities appeared to be found in an enzyme which had not been previously explored fully in its effect on burn eschar. This enzyme is the Bromelains\* derived from the stem and leaf of the — pineapple.<sup>9,10</sup> The author obtained the Bromelains for investigational use on animals. The material consists of a dry powder which is described in its pharmacological and biochemical properties by Smyth, Moss, Frazer and Martin.<sup>32,33,35,48,49</sup> The Bromelains is a proteolytic enzyme. In its dry consistency it is stable at room temperature when occluded from humidity and oxygen. It is active by the addition of water, but undergoes autolysis slowly and consequently should be used within a reasonable time.

In the summer of 1963 the effect of this Bromelain on human eschar,\*\* obtained by sharp debridement from a severely burned patient, was investigated at the Chemistry Department of a College by the author with Mr. Samuel W. Cushman, who utilized part of this work for his undergraduate honors thesis.<sup>47</sup> In the test

\*Rorer Pharmaceutical Grade Bromelains supplied by William H. Rorer, Co., Inc., Fort Washington, Pennsylvania.

\*\* Regional Memorial Hospital, Brunswick, Maine.

tube, human eschar was exposed to different concentrations of Bromelains at different temperatures and under different gasses. The experiment showed a rapid disruption in the continuity of the human eschar by the enzyme in every concentration and was faster when oxygen was excluded. Good debriding action was obtained at body temperature.

Following this, the enzyme was applied to experimental burns on rabbits in small chambers for testing the activity in different environments. Again, elimination of oxygen seemed to accelerate the debriding process. Of major importance was constant contact of the enzyme with the eschar. This was obtained by spreading the powder in a thin layer on the eschar, covering it with a layer of agar, and then sealing it with a plastic sheet. In this way, deep third degree burns on rabbits were debrided in less than six hours, without demonstrable damage to viable tissue for this length of exposure.

After the rapidity and selectiveness of the debriding properties of the Bromelains were proved, an attempt was made to show that the so-exposed wound-bed would accept a graft, and that there were no toxic side-effects to the organism.

An experimental full thickness third degree burn was produced on an anesthetized piglet by metal preheated to a certain temperature and placed with known weight for a known length of time on shaven and cleaned skin (Fig. 1). In order to simulate the clinical requirements of postponing debridement of a severely burned human until shock was overcome and the fluid and electrolyte balance established, the piglet was left untouched for two days. The Bromelains powder was then applied to the burned surface in a quantity of 10 mgs. per sq. cm., and covered with an agar paste composed of 3 grams of agar to 100 milliliters of normal saline. It was attempted to have about 1 c.c. of agar covering 1 sq. cm. of burn. This agar sheet secures the active Bromelains in unchanged contact with the eschar. Because of the restless nature of the piglet, which was not kept under anesthesia during the whole debriding time, it was necessary to lay a protective rubber ring around the treated area and then seal it from oxygen by a plastic sheet. After two hours the agar and Bromelains powder were removed, and the result is seen in Fig. 2. There was a considerable softening of the previously firm eschar. The same process of Bromelains application was repeated, and, after another two hours, marked debridement was visible (Fig. 3). After a third two-hour Bromelains application, the entire third degree full thickness burn was debrided without any sign of damage to viable tissue or bleeding (Fig. 4). During this whole process the piglet showed no clinical signs of toxicity. Following the accepted concept of preparing a debrided burn surface for grafting, the wound bed was treated with normal saline soaks for another 48 hours (Fig. 5). A fine whitish film which had remained on the base of the wound had not been affected by the

process, and it was thought better to remove it by scraping before grafting. This was done with negligible bleeding. The surface so prepared (Fig. 6) was then covered with a split-thickness skin graft (Fig. 7) and dressed. One week after this grafting procedure, the graft had fully taken (Fig. 8) and remained viable with no contractions. The piglet showed no signs of organic damage.

The same experiment was repeated on several other piglets with the same results. In some of the experiments the burn surface after the complete debridement was kept exposed to the enzymes for another 48 hours indicating that even after prolonged exposure viable tissue was not attacked by Bromelains.

#### SUMMARY

This Bromelains powder, when hydrated and kept in contact with the eschar by agar paste, and sealed from oxygen by plastic sheeting, has debrided a full thickness third degree experimental burn on piglets in six hours. Proteolytic activity occurred to devitalized tissue only. There was negligible blood loss. No toxic side effects were observed in the piglets. A split-thickness skin graft took completely. The preparation and application is simple and the enzyme is inexpensive. These qualities seem to meet the requirements for an early debriding agent.

This has been an encouraging experience. However, further investigation must be undertaken to prove the same effectiveness on human eschar and the non-toxicity of the agent in humans. In addition, animal experiments have to be continued to investigate the optimum time of debridement following the burn, the effect of the enzyme on burns of a different depth, the effect of the enzyme debriding action by pre-treatment of the eschar with different agents and improvement of the technique of application.

These investigations are now under way and are thus far encouraging. They will be reported in a subsequent publication.

This work, done while carrying on a busy practice, was made easier and more pleasant by the cooperation of Mr. Samuel W. Cushman, Dr. S. Kamerling and Dr. John C. Houck, Ph.D.

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FIGURE 1  
Three-week-old piglet, 2 days post burn, pre-debridement.

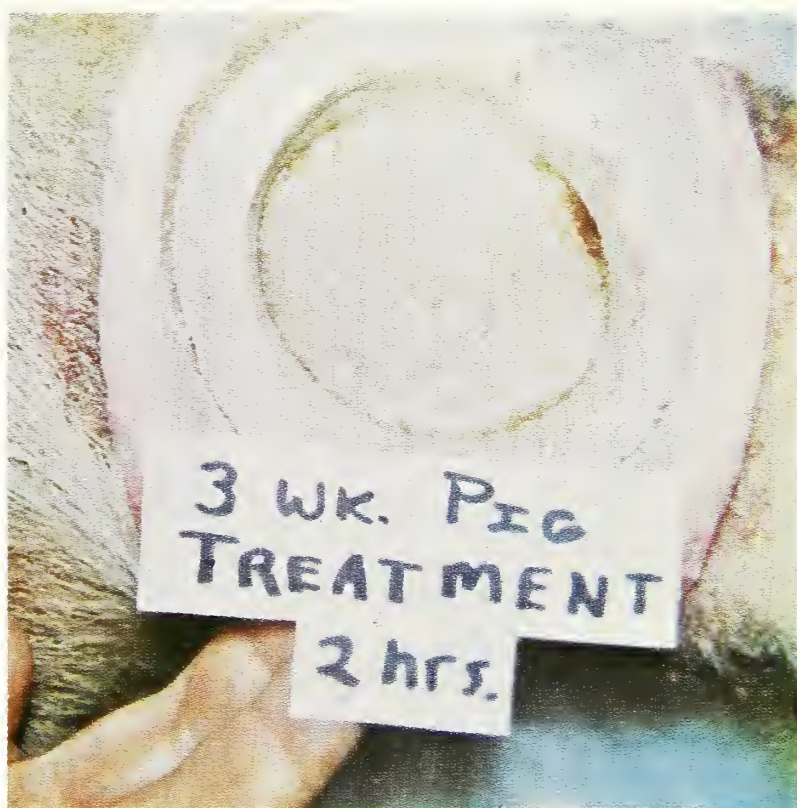


FIGURE 2  
Same burn wound after 2 hours debridement with Bromelain-Agar.

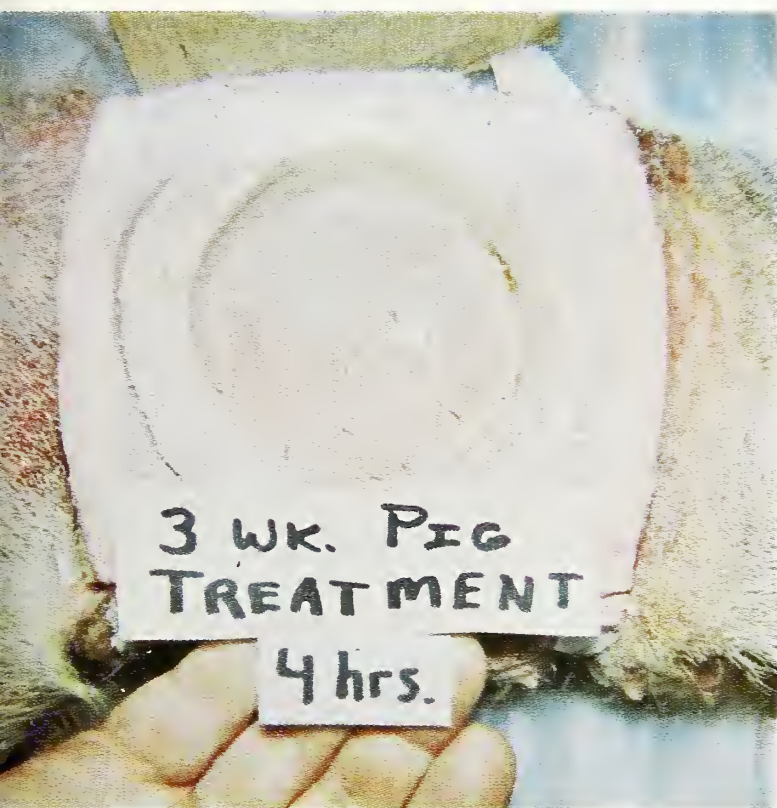


FIGURE 3  
Same burn wound after 4 hours debridement with Bromelain-Agar.

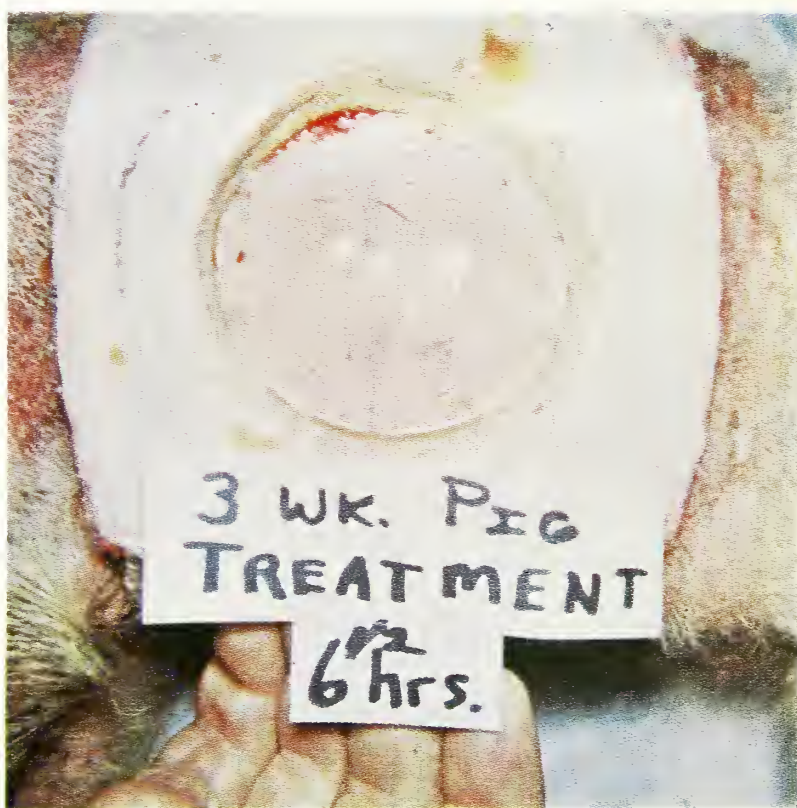


FIGURE 4  
Same burn after 6½ hours debridement with Bromelain-Agar.





FIGURE 5  
Same burn wound without chamber after 6½ hours  
debridement with Bromelain-Agar.

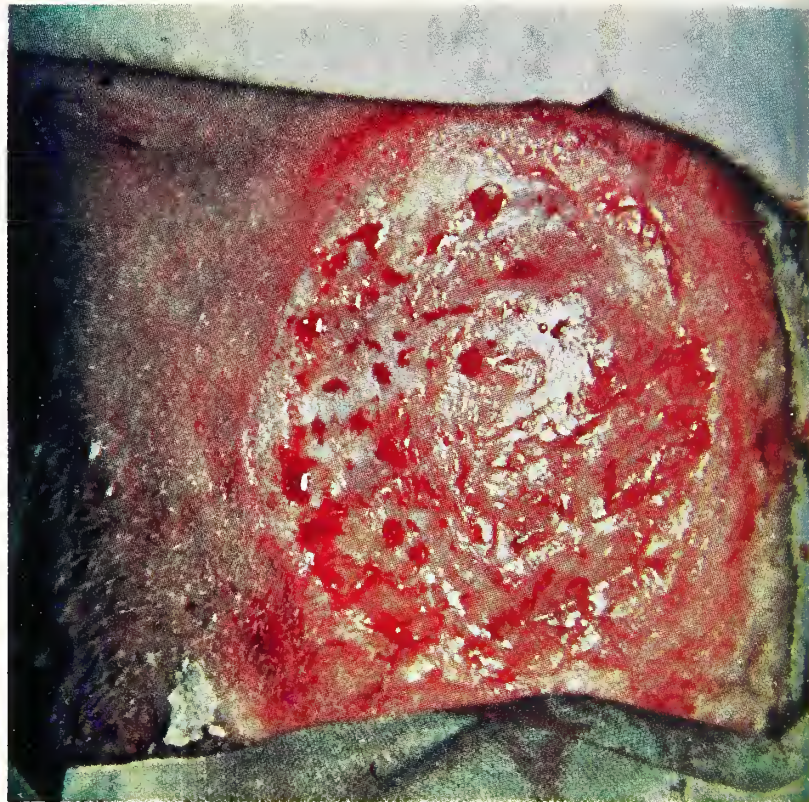


FIGURE 6  
Same burn wound after scraping pre-grafting 2 days  
after debridement.



FIGURE 7  
Same burn wound immediately after grafting.



FIGURE 8  
Same burn wound 1 week after grafting.



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56 Baribeau Drive, Brunswick, Maine

# Postgraduate Medical Education in Maine: A New Experiment Utilizing Two-Way Radio and Television†

FRANK M. WOOLSEY, JR., M.D.\* AND WILLIAM T. STRAUSS, M.D.\*\*

The increasingly rapid development of new medical knowledge, accompanied by the necessity for the practicing physicians to keep abreast of such developments, makes it imperative for the doctor to extend his medical education on a continuing basis in order to provide his patients with optimal care. Although this is true in all localities, the opportunities for obtaining postgraduate medical education vary widely in different states and communities. Physicians in certain areas which are in close proximity to medical schools find it relatively easy to take advantage of special courses, lectures, seminars, etc. However, there are many other regions, such as the State of Maine, which have no medical schools or centers of postgraduate training to which physicians can conveniently go for their continuing education. Such physicians of necessity must rely on the written word to a large extent, in addition to relatively infrequent attendance at formal courses, etc.

A unique approach to a partial solution of this problem was started in 1955 by the Department of Postgraduate Medicine of the Albany Medical College, which developed the first use of two-way radio conferences. Space does not permit a full description of how this system operates and the reader is referred to several reports for complete details.<sup>1-6</sup> Briefly, however, as used by the Albany Medical College, the two-way radio communication concept refers to teachers in a medical school presenting educational materials by radio to physicians in community hospitals and the latter directing their questions and discussions by radio to the teachers. This is effected through the utilization of non-commercial FM radio broadcast transmitters fed by the medical college studio\* and by transmitters located in community hospitals. In addition, all participants view materials sent to the participating hospitals in advance of the program. These color slides, ECG tracings, roentgenograms, etc., are shown on cue by the faculty. During each conference, twelve widely separated hospitals can effectively participate in easy discourse. The questions and discussions originating in these remote participating groups are heard by everyone tuned to the broadcasting station and result in effective inter-communication. In the Albany Medical College Network, which now consists

of approximately 65 community hospitals and more than 15 medical schools, the ease of communication and the integration and coordination of thought and material is further enhanced through a tone network alerting system which allows the local hospital moderator in any one of the participating groups to turn on his own individual light in front of the medical college moderator by simply pressing a button on the front panel of his transmitter. Through this arrangement the program moderator at the medical college knows immediately when someone wishes to ask a question or enter the discussion and there is complete continuity of questions, discussions, and answers.

From the above abbreviated description it is at once obvious that this technique is quite different from one-way radio broadcasting. The effects, too, are quite different since the participants in such a system receive vastly more pertinent information from the two-way broadcasts than if they were only able to listen and not participate. Not only can they ask questions and receive answers, but they can also hear the queries and discussions of their peers, a situation which approximates the classical setting of the classroom.

Some physicians in Maine are already acquainted with this system since hospitals in Portland and Lewiston participated in the one-hour weekly programs from October 1963 to April 1964. Beginning September 30, 1964 three or four additional hospitals in Maine (Bangor, Waterville, Augusta and possibly Presque Isle) will become network participants.

The unique feature of this season's programs is that they will be broadcast on open circuit by several educational *television* channels to be announced next month.

The audio portion of the program will be identical to that in the two-way radio system, as will much of the video portion. However, the television screen will carry additional visual material when the participating hospital audience is not viewing the instructional slides.

The great advantage of adding television to the two-way system is that physicians in non-participating hospitals and in their homes and offices can now take advantage of this form of instruction. Their questions too, can be answered since "non-participating" viewers need only telephone their queries to one of a number of points located throughout the state whence they will be relayed to the nearest participating hospital for insertion into the two-way radio and television broadcast. The answer will be received via the television system.

†From the Department of Postgraduate Medicine, Albany Medical College of Union University.

\* Associate Dean, Professor of Postgraduate Medicine and Chairman, Department of Postgraduate Medicine, Albany Medical College of Union University.

\*\* Assistant Professor of Postgraduate Medicine, Albany Medical College of Union University.

\*WAMC-FM



The staffs of all participating hospitals in Maine will be fully informed at staff meetings about this important advance in continuing medical education. These programs will be brought to you by the State of Maine Educational Television Network and other stations to be announced later in cooperation with the Maine Medical Association and the Department of Postgraduate Medicine at Albany Medical College of Union University.\* All Maine physicians will receive detailed information about the programs, by mail from the Executive Director of the Maine Medical Association shortly after September first.

This concept is a "first" in postgraduate medical education and it is hoped that all Maine physicians will take advantage of this new method of assisting them with their continuing medical education. These two-way

conferences will be presented from 12 noon to 1:30 P.M. on Wednesdays, beginning on September 30.

\*This department at present consists of the following in addition to the authors: William P. Nelson, III, M.D., Assistant Dean, Professor of Postgraduate Medicine; Salvatore Tabacco, M.D., Instructor in Postgraduate Medicine; Henry S. M. Uhl, M.D., Assistant Professor of Postgraduate Medicine; Albert P. Fredette, Station Manager, WAMC-FM.

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Dr. Strauss, 47 New Scotland Avenue, Albany, New York

## SURGICAL TREATMENT OF MASSIVE PULMONARY EMBOLIZATION — REPORT OF A CASE

*Continued from Page 168*

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Dr. Drake, 18 Bramhall St., Portland, Maine  
Dr. Rand, Maine Medical Center, Portland, Maine  
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Dr. Dillihunt, Maine Medical Center, Portland, Maine

## TUBERCULOSIS — GOING, GOING . . .

As a result of widespread use of the anti-tuberculous drugs, the mortality from tuberculosis has decreased sharply, and the length of hospital stay and the relapse rate have also greatly decreased. Of all the people who have ever lived, more have died of tuberculosis than of any other disease. Our generation has been fortunate to have been given the tools with which this once dreaded disease may be eradicated. Let us not fail to use them wisely. — A. C. Cohen, M.D. in **Pennsylvania Medical Journal**, 67:2 (Feb.) 1964.

## Boston Medical Reports

MASON TROWBRIDGE, JR., M.D.

A second review of the recent medical television series (requested by the B.M.R. group) will necessarily be somewhat redundant. My conclusion in the previous review was that television lectures without audience participation would do little to raise standards of medical care. Only a small fraction of the medical community heard the programs. And, as with church, those that needed it most weren't there. The failure of TV to get across a message to a significant percentage of the medical community has been reported elsewhere.

Indignant statements that the series was directed towards second year medical students were often heard. The B.M.R. group had at least one warning in advance to make the presentations reasonably sophisticated. Too often a cardiologist who gives excellent presentations at Boston meetings tells a Bangor audience not to digitalize a patient merely because he has a murmur. The speaker has an obsessive compulsion not to "talk over anyone's head" but has no hesitation about boring the more astute members of the audience. Any teaching program in an area should be directed to the latter group, since local medical education is achieved by the activities of these men at conferences and in consultations. One program which was elementary and excellent was that of Dr. Schwartz and Dr. Relman. Simple routine urinalysis is indeed a stepchild of medicine.

The medical TV speaker has two strikes on him to start with, since without an audience his presentation is almost universally wooden. The tapes of the California Medical Society are far more worthwhile; it is apparent that the speaker is addressing a responsive audience. The tapes can be run when one feels in the mood, can be re-run if one misses a point, and are inexpensive. They are a poor substitute for reading, but open new vistas and lead to further study.

The previous review stressed the fact that university teachers have a considerable obligation not only to sow the seeds of wisdom but to make certain that their seeds are falling on well prepared ground. University teachers rarely admit that they have any responsibility for maintaining the receptive soil. Everyone mouths the cliché that the family doctor (internist or general practitioner) is the keystone of good medical care in a community. As an internist, I have had a thorough seasoning in the minor leagues starting with practice in Lubec,

Maine. I read with interest the many articles that question whether the family doctor can survive in our society. University physicians are vaguely aware of this problem but do not heed Freymann's<sup>1</sup> advice to leave the ivory towers more often and participate in the fray.

Didactic lectures to physicians harried by the problems of private practice are of dubious value. The lives of physicians in the hinterlands are probably no more hectic than in Boston where at least one hospital has staff meetings at breakfast. Late one night several physicians were passing up the B.M.R. program to dictate summaries. University physicians are vaguely aware of this unfortunate situation. One thing they can do to correct it is to beef up their departments of public health, now often called departments of community health. These are concerned with increasing the productivity of the individual physician without increasing wear and tear on his adrenals. The only major organization concerned with the gradual coarctation of the aorta of internal medicine is the American Society of Internal Medicine. Some University internists do not support it, feeling that the leak is not in their end of the boat.

It is hard to state whether the B.M.R. reports were worthwhile since I do not know how much money and energy went into their production. The Boston group has a better idea than I as to the number of doctors listening. The Bangor Medical Club has an informal bimonthly telephone and loudspeaker tieup with the Massachusetts General Hospital. In Jimmy Durante's immortal words, "Everybody is trying to get into the act." A doctor in Bangor gives a paper that has previously been sent to Boston. Discussion of the paper, occasionally becoming an intellectual roughhouse, ensues. When this experiment is written up we will report the ergs of energy expended by Boston participants, the cost (\$16 for a lot of conversation), the logarithm of the number of synopses activated in Bangor, and will apply the chi square formula to the whole experiment. Without such data a report on an educational experiment is of dubious value.

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142 Pine Street, Bangor, Maine





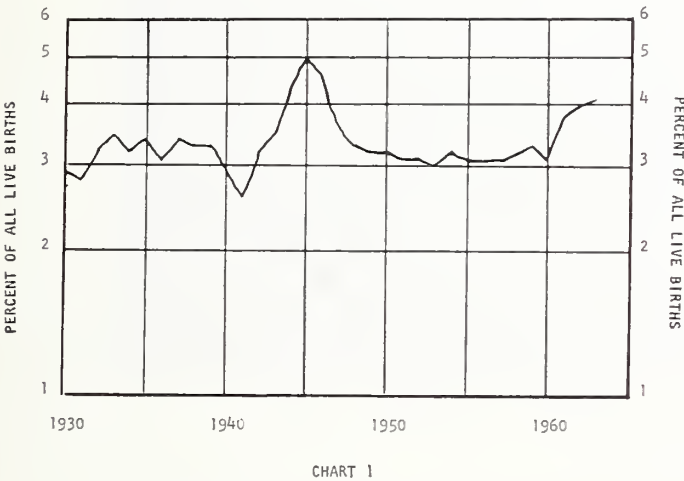
DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine  
Department of Health and Welfare

Illegitimate Births in Maine, 1963

EDSON K. LABRACK, M.P.H.\* and DALE E. WELCH\*\*

In 1963 there were 901 children born out of wedlock to mothers who were residents of Maine. These births represented 4.1% of the 21,919 resident live births during the year. The proportion of illegitimate births in 1963 was up slightly over the proportion in 1962. This marks the third successive year during which the proportion of illegitimate births has risen. Trends in illegitimate births between 1930 and 1963 are shown in Chart 1.



TREND IN ILLEGITIMATE BIRTHS

During the middle and late 1930's the proportion of illegitimate births remained relatively stable around 3.3%. In 1940 the proportion dropped sharply, but during World War II it rose to a peak of 5.0% in 1945. After the war the percentage dropped rather sharply and during the 1950's was quite stable around 3.1%. In 1961 the proportion started to rise and has risen steadily during each of the past 3 years. The phenomenon of an increasing proportion of illegitimate births is not peculiar to Maine. Data available for 38 states show an increase between 1960 and 1962 in approximately the same proportion as the increase in Maine.

Comparisons with U. S. illegitimate births in this report concern U. S. white illegitimate births only, since less than 1.0% of the population of Maine is nonwhite.

\*Director, Division of Research and Vital Records.  
\*\*Statistician, Division of Research and Vital Records.

TABLE 1

Total resident live births and resident illegitimate live births by counties. Maine, 1963

	Total live births	Illegitimate live births	Percent
STATE	21,919	901	4.1
Androscoggin	1,943	77	4.0
Aroostook	2,750	65	2.4
Cumberland	4,129	200	4.8
Franklin	473	27	5.7
Hancock	632	25	4.0
Kennebec	1,919	89	4.6
Knox	524	23	4.4
Lincoln	365	12	3.3
Oxford	954	26	2.7
Penobscot	3,239	151	4.7
Piscataquis	338	11	3.3
Sagadahoc	519	30	5.8
Somerset	871	32	3.7
Waldo	497	28	5.6
Washington	585	42	7.2
York	2,181	63	2.9

GEOGRAPHIC DISTRIBUTION

Table 1 shows the number and percent of illegitimate live births in each county in Maine. The proportion of illegitimate births ranged from a low of 2.4% in Aroostook County to a high of 7.2% in Washington County. The proportion of illegitimate births were significantly lower in Aroostook, Oxford, and York Counties and higher in Washington County than the proportion in the remainder of the State. Differences exhibited by other counties were within the normal range.

AGES OF MOTHERS

The median age of mothers giving birth to a child born out of wedlock was 21.4 years. Percentage-wise, illegitimate births were at their highest in females under 15 years of age. However, only 22 of the 21,919 chil-

Continued on Page 180

## *Maine Heart Association Notes*



### **Therapy of Severe Rheumatic Carditis**

“. . . extensive data collected at the House of the Good Samaritan have shown that steroids in large dosage cause a considerably higher incidence of regression and disappearance of significant murmurs than does aspirin. . . .

“. . . This conclusion is evident from the markedly lower death rate and from the faster and higher recovery rate in our steroid-treated group. . . . Appearance or worsening of congestive heart failure occurred also much more rarely during steroid therapy than during aspirin treatment.

“In spite of the fact that treatment in this series could not be determined by random selection, the observations collected . . . strongly support our clinical experience that steroids in large dosage are the treatment of choice in severe rheumatic carditis with congestive heart failure or pericarditis.”

---

Reference: Czoniczer, G., M.D., et al. *Circulation*, Volume XXIX, No. 6, pages 813-819, 1964.

---

### **American Heart Association's Scientific Sessions and Annual Meeting**

The 37th Scientific Sessions and annual meeting will be held at Convention Hall, Atlantic City, N. J., October 23-27, 1964. The Scientific and Clinical Sessions are scheduled for October 23-25 and will comprise six half-day meetings designed to meet the needs of the practicing physician. The Clinical Sessions are designed as a post graduate course in cardiology.

Simultaneous sessions are scheduled for the presentation of papers based on investigative work related to the cardiovascular field.

The Annual Meeting will be on October 25-26-27 at the Chalfont-Haddon Hall.

An extensive program of special events is scheduled for the Ladies. For advance Registration write the Maine Heart Association, 116 State Street, Augusta, Me., or the American Heart Association, 44 East 23rd Street, New York, N. Y., 10010.





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**"Diverticulitis** Mild, chronic symptoms of diverticulitis, such as diarrhea or flatulence also are treated<sup>1</sup> by low-roughage diet, adequate fluid intake and Metamucil. . . ."

Usual Adult Dosage: One rounded teaspoonful of Metamucil (or one packet of Instant Mix Metamucil) in a glass of cool liquid one to three times daily.

Metamucil is available as Metamucil powder in containers of 4, 8 and 16 ounces and as flavored Instant Mix Metamucil in cartons containing 16 and 30 single-dose packets.

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**SEARLE**

*Research in the Service of Medicine*



# *From the Secretary's Notebook*

## 111th Annual Session of the Maine Medical Association House of Delegates

The 111th annual meeting of the House of Delegates of the Maine Medical Association was held on Sunday, June 14, 1964 at Rockland, Maine with sixty members present. The first meeting was called to order at 9:45 A.M. by the President-elect, Thomas A. Martin, M.D., who turned the meeting over to the Speaker of the House, Linus J. Stitham, M.D. This meeting adjourned at 12:20 P.M. The second meeting of the House was called to order at 3:00 P.M. and adjourned at 5:15 P.M.

### **Election of Speaker and Vice-Speaker**

Linus J. Stitham, M.D. was reelected Speaker of the House of Delegates and Charles R. Glassmire, M.D. was elected Vice Speaker of the House.

### **Amendment to the Constitution and By-Laws of the M.M.A.**

The following recommended amendment to the Constitution, as proposed by the Penobscot County Medical Association to eliminate the Fall Clinical Session, was approved:

Constitution, Article VII, Section 2, "Meetings of the Association may be called by the President, or by the Council, and shall be called by the President on petition of ten (10) members of the House of Delegates or fifty (50) members of the Association. There shall be more than one (1) scientific meeting sponsored by the Association each year."

PROPOSED AMENDMENT: "That the Constitution of the Maine Medical Association, Article VII, Section 2, be changed so that the Association is not required to sponsor more than one Scientific Meeting each year." The last sentence of the article would be changed to read: There shall be one (1) scientific meeting sponsored by the Association each year.

(This proposed amendment was presented at the Interim Meeting of the House of Delegates, published in the May issue of The Journal and copy sent to each member of the House with Summary of the Interim Meeting.)

It was pointed out that this does not preclude the setting up of any other scientific session. It merely changes the Constitution so that it is not necessary to have a Fall Clinical Session.

### **Reference Committees**

The following Reference Committees were appointed by the Speaker of the House, Dr. Stitham:

Reference Committee No. 1 – George W. Wood, III, M.D., Chairman; John F. Gibbons, M.D. and Roger J. P. Robert, M.D.

Reference Committee No. 2 – Peter B. Aucoin, M.D.,

Chairman; Ralph C. Powell, M.D. and Samson Fisher, M.D.

Reference Committee No. 3 – Karl V. Larson, M.D., Chairman; John B. Madigan, M.D. and Russell M. Lane, M.D.

### **A. H. Robins Community Service Award**

This award was presented to Charles W. Steele, M.D. of Lewiston.



Dr. Stein, President of the Maine Medical Association, presents the Community Service Award to Dr. Steele.

### **Annual Reports**

Reports were submitted prior to the meeting by the following and included in the House of Delegates' folder:

John F. Dougherty, M.D., Councilor and Council Chairman, and Councilors, Paul S. Hill, Jr., M.D., Charles F. Branch, M.D., George E. Sullivan, M.D., Raymond E. Weymouth, M.D. and Clyde I. Swett, M.D.

Standing Committee Chairmen: Recruitment, Aid and Placement, Paul H. Pfeiffer, M.D.; Rural Health, George W. Bostwick, M.D. and Health Insurance, Francis A. Winchenbach, M.D.

Special Committee Chairmen: Amy W. Pinkham Fund, Norman H. Nickerson, M.D.; Diabetes, Melvin



Bacon, M.D.; Maternal and Child Welfare, Alice A. S. Whittier, M.D.; Mental Health, Guy N. Turcotte, M.D.; Disaster Medical Care, John T. Konecki, M.D.; Blood Transfusion, Joseph E. Porter, M.D.; Liaison Activities between the Maine State Nurses' Ass'n. and the M.M.A., George O. Chase, M.D.; Advisory Committee to the Secretary of State and to the Bureau of Motor Vehicles, George L. Maltby, M.D.; Maternal Mortality, Robert M. Knowles, M.D.; Medicine and Religion, Asa C. Adams, M.D. and Rehabilitation, John J. Lorentz, M.D., and by the Secretary-Treasurer, Esther M. Kennard.

Reports were presented at the meeting by the Executive Director, Daniel F. Hanley, M.D. and by the following committee chairmen: Brinton T. Darlington, M.D., Legislative; Thomas A. Martin, M.D., Medical Advisory; Joseph E. Porter, M.D., Blood Transfusion; and Peter W. Bowman, M.D., Problems on Long-Term Patient Care.

#### Board of Directors — Associated Hospital Service

It was voted to reduce by one the number of M.D.'s on this Board to bring the Board into a 7-7-7 split — seven lay members, seven physicians and seven members of the Hospital Association.

#### Maine Medical Education Foundation

It was voted that the Foundation request this year be sent out as a \$25.00 assessment rather than a voluntary contribution. This is for one year.

#### Group Malpractice Insurance for M.M.A. Members

Urged the committee which has been set up by the Council to continue investigating the possibility of securing group malpractice insurance for M.M.A. members.

#### Report of Committee on Blood Transfusion

Recommended that a summary of this report be sent to each hospital and each county society.

#### Report of Committee on Maternal Mortality

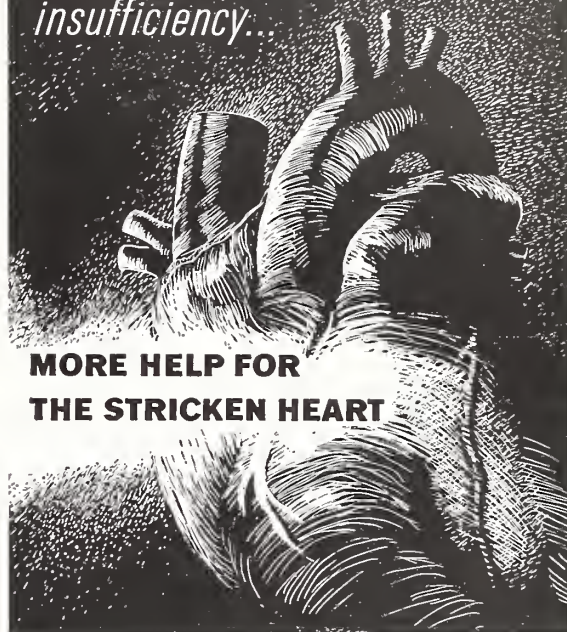
It was voted that death certificates be changed to include a check box in which to indicate whether pregnancy has occurred within 90 days. It was recommended that maternal death committees be set up in each hospital and that these committees be instructed to report to the State committee on maternal deaths. The recommendation to broaden the membership of the State Maternal Death Committee geographically was approved.

#### Report of Maternal and Child Welfare Committee

This report stated that a new federal grant will be available to Maine through Maternal and Child Health for a special project of comprehensive maternal and infant care. It was voted that the committee contact Dr. Hanley in regard to this grant and, if he is in favor, to sponsor the drawing up of a plan with the approval of representative obstetricians, pediatricians and general practitioners. It was voted to endorse the PKU program

*Continued on Page 182*

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treatment  
of your patients  
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DEPARTMENT OF HEALTH AND WELFARE — Continued from Page 175

TABLE 2

Illegitimate live births and birth rates by age groups: Maine, 1963 and 1958, and U. S. estimated<sup>1</sup> 1963.

Age	Maine		1958		U.S.
	1963		1958		1962
	Illegitimate	Rate per	Illegitimate	Rate per	Estimated
	births	1,000 <sup>2</sup>	births	1,000 <sup>2</sup>	rate per 1,000
Total	901	3.8	741	3.3	2.3
10-14	15	0.3	11	0.3	0.2
15-19	357	8.4	275	8.3	5.5
20-24	286	8.6	239	8.5	6.0
25-29	107	3.2	97	3.3	2.5
30-34	92	3.1	76	2.5	1.4
35-39	32	1.0	32	1.0	0.7
40+	12	0.4	11	0.4	0.2

1. Source: *Vital Statistics of the U.S.*, 1962, Vol. 1, U. S. Department of Health, Education and Welfare.
2. Illegitimate live births per 1,000 females in specified age group.

TABLE 3

Total live births, legitimate and illegitimate live births and percentage of illegitimate births by age of mother: Maine, 1963, and estimated percent of white illegitimate births, 1962.

Age of mother	Total live births	Illegitimate births	Percent illegitimate	Estimated U.S. 1962
Total	21,919	901	4.1	2.8
Under 15	22	15	68.2	48.3
15-19	3,249	357	11.0	7.9
20-24	7,929	286	3.6	2.7
25-29	5,336	107	2.0	1.4
30-34	3,232	92	2.8	1.3
35-39	1,659	32	1.9	1.5
40 and over	492	12	2.4	1.8

dren born to residents of Maine in 1963 were born to mothers under 15. Females aged 15-19 years were responsible for the largest number of illegitimate births. Three hundred and fifty-seven or nearly 40% of all illegitimate births occurred in this age group, and 11% of all children born to mothers 15-19 years old were born out of wedlock. However, the age group most susceptible to illegitimate birth was the 20-24 age group. While the proportion of illegitimate births was lower (3.6% of all live births), the illegitimate birth ratio was 8.6 per 1,000 women aged 20-24 years of age as compared with 8.4 per 1,000 women aged 15-19 years. National data and data from a 1958 Maine study show similar relationships.

The seeming inconsistency in these data is due to the fact that there are nearly 10,000 more females in the State in the 15-19 year age group than in the 20-24 year age group and the fertility rate of 20-24 year old females is over 3 times as great as that of 15-19 year olds.

Table 2 shows age specific illegitimate birth rates for females of specified ages in Maine in 1963 and 1958 and estimated rates for white females in the U. S. as a whole in 1962. The rate of illegitimate birth was

TABLE 4

Illegitimate births by age and number of children born to Mother: Maine, 1963.

Age of mother	Children born to mother prior to this birth						
	Total	0	1	2	3	4	5 or more
Total	901	531	110	74	52	51	83
Under 15	15	14	1	—	—	—	—
15-19	357	320	30	6	—	1	—
20-24	286	168	47	36	20	9	6
25-29	107	9	12	16	19	19	32
30-34	92	16	13	10	6	14	33
35-39	32	4	6	2	6	6	8
40 and over	12	—	1	4	1	2	4

higher in Maine in all age groups than in the U. S. as a whole. Differences were statistically significant at all age groups between 15 and 34 years.

Comparison of 1963 data with 1958 data indicates that about 80% of the increase in the number of illegitimate births between the two years can be attributed to an increase in the number of children born out of wedlock to mothers 15-24 years of age. There were 129 more children born out of wedlock to women aged 15-24 years of age in 1963 than in 1958. The increase, however, is not due to any increase in rate of illegitimate births to women at these ages, but rather to an increase in the number of these women in the population of the State as children of the so-called "post war baby boom" reach childbearing age. During the 5 year period between 1958 and 1963 the estimated number of females aged 15-19 years increased by about 9,200 (from 33,100 to 42,300) and the number of females aged 20-24 years increased by about 4,900 (from 28,200 to 33,100).

The increase in the percent of illegitimate births during the past 5 years may be attributed to a population increase in the number of females in the State aged 15-19 years during this period. This age group has a relatively low birth rate, but a high percentage of births in this age group are illegitimate. The number of females in this age group is expected to increase for another 2 or 3 years, and during this period it is likely that the overall percent of illegitimates will continue to rise, then it may be expected to decline as the size of the 15-19 age group stabilizes and the size of the 20-24 age group, with its high fertility rate and lower percentage of illegitimates, starts to increase in size.

It is important to note that the increase appears to be due to the increase in the number of females of childbearing age. There has been no significant change in the rate of illegitimate births in any age group during the 5 year period 1958-1963 (See Table 2).

PARITY OF MOTHERS OF ILLEGITIMATES

About 60% of illegitimate births to residents of Maine in 1963 were first births to the mother. There were 30



women who had borne 9 or more children prior to the illegitimate birth and one woman was bearing her 16th child. Table 4 shows parity statistics for women who gave birth to a child born out of wedlock in Maine in 1963.

#### SUMMARY AND CONCLUSIONS

Illegitimate births increased in number and in percent

of all live births for the third successive year in 1963. The increase in illegitimate births was due to an increasing number of females in the State reaching childbearing age. There was no evidence which indicated that the increase was due to factors other than population increase in women of childbearing age. The rate of illegitimate births in 1963 was not significantly different from the rate 5 years ago before the increase began.

## Miss Nash Retires

After 18 years of service as Director of the Division of Hospital Services, Lillian Nash, R.N. retired from the Department on July 1. She came to serve in this capacity upon resignation from the Gardiner General Hospital, Gardiner, Maine in September, 1946 after 4 years of service as superintendent. She served formerly as head of the institution from 1924 to 1929 and was also an instructor of nurses at the Knox General Hospital in Rockland for several years. She received her professional training at the Gardiner Hospital with affiliation at the New Haven Hospital in New Haven, Connecticut. Following, she did graduate work at the Massachusetts Eye and Ear Infirmary and the Providence Lying-In Hospital. She is a graduate of the Eastern State Normal School at Castine and also attended Simmons College.

During her years of devoted service with the Department, Miss Nash made an enviable record for herself in the promotion of high standards of nursing care

throughout the services under her jurisdiction as well as all others with which she was allied. This was especially true of the nursing home field. Her early leadership in this direction was largely instrumental in bringing about the present Maine State Nursing Home Association. A member of the Maine Hospital Association as well as similar professional organizations, she assisted continuously in helping to stimulate the best possible conduct of nursing care everywhere in the State and region. She carried her heavy administrative duties with poise and dignity always and was unfailing in her effort to bring them to a high degree of professional performance.

Miss Nash is replaced by William J. Carney, formerly health officer for the City of Bangor, who has accepted these duties in addition to those he is currently carrying on in conjunction with his position as Public Health Advisor on State assignment from the U.S. Public Health Service.

## Announcement

### Central Maine General Hospital, Lewiston, Maine Fall Postgraduate — Refresher Course in Hematology Beginning Wednesday, September 30, 1964

Wednesday, September 30 — IMMUNOGLOBULINS.

Robert S. Schwartz, M.D., Assistant Professor of Medicine.\* This lecture will include consideration of the Thymus and Auto-Immune Diseases.

Wednesday, October 7 — POLYCYTHEMIA AND ERYTHROCYTOSIS.

W. Jack Mitus, M.D., Senior Instructor in Medicine.\* This lecture will include discussion of all Myeloproliferative Diseases.

Wednesday, October 14 — LEUKEMIA.

William Dameshek, M.D., Professor of Medicine.\* This lecture will include all aspects, including newer modes of therapy.

Wednesday, October 21 — IDIOPATHIC THROMBOCYTOPENIC PURPURA.

Mario Baldini, M.D., Associate Professor.\* This discussion will include other forms of Purpura and Thrombocytopenia.

Wednesday, October 28 — HEMORRHAGIC DISORDERS.

Walter Tannenberg, M.D., Instructor in Medicine.\* This discussion will be on the whole general subject with emphasis on diagnosis and treatment.

\*Tufts University School of Medicine.

This course will total 15 hours. Application has been made to the American Academy of General Practice for credit under Category 1.

All lectures will be held in Hiebert Hall, Central Maine General Hospital from 3:00 to 6:00 p.m. Fee for the course is \$30.00.

FROM THE SECRETARY'S NOTEBOOK  
*Continued from Page 179*

and also the State Vision Testing program for three-year-old children.

**Nominating Committee Report**

The report of the Nominating Committee, consisting of Standing Committees for 1964-1965 was accepted. This report was published in the July, 1964 issue of The Journal of the Maine Medical Association – page 138.

**Budget for 1965**

George E. Sullivan, M.D., Chairman of the Budget Committee, reviewed the proposed budget for fiscal year 1965 as approved by the Council and presented at the Interim Meeting of the House of Delegates. On motion duly made and seconded, the budget was approved.

Estimated income from January 1, 1965 to December 31, 1965 from State Dues, Journal Advertising, Subscriptions, Exhibit Space Rentals and miscellaneous is \$62,625.00.

Approved expenditures are itemized below:

Association	
Office	
Salaries:	
Executive Director	\$11,000.00
Secretary-Treasurer	3,500.00
Stenographers	7,000.00
Travel — Exec. Dir. & Secy.-Treas.	1,200.00
Supplies, tel., rent, Payroll taxes	5,500.00
Equipment	500.00
General:	
President's Expenses	1,000.00
Annual Session & Interim	
Meeting House of Delegates	5,000.00
Council	300.00
Committees:	
Med. Advisory (Legal Counsel)	1,000.00
Legislative Counsel	1,500.00
Standing & Special	1,500.00
Delegates:	
American Medical Assn.	1,200.00
N. E. & New Brunswick	400.00
New England Council Dues	150.00
Fall Clinical Session	500.00
Annual Rosters	450.00
Woman's Auxiliary	400.00
Journal:	
Printing & Plates	15,000.00
Travel	100.00
Office	
Salaries:	
Editor	2,500.00
Secretary-Treasurer	3,000.00
Stenographer	3,500.00
Supplies, postage, rent	
Payroll taxes	1,800.00
Insurance	100.00
Retirement Fund	2,400.00
Totals	\$70,500.00

**Election of Councilors**

James C. Bates, M.D. of Eastport was elected Coun-

cilor for the Fifth District which includes Hancock and Washington counties, and Asa C. Adams, M.D. of Orono, Councilor for the Sixth District, including Aroostook, Penobscot and Piscataquis counties.

(Following the meeting, delegates of the Lincoln-Sagadahoc and Knox County Societies elected Edward K. Morse, M.D. of Rockland as Councilor for the Third District for one year to fill the unexpired term of Dr. Dougherty, who was elected President-elect at the General Assembly on Monday, June 15, 1964.)

The stenographic record of this meeting of the House of Delegates is on file in the Association's headquarters where it is available to any member of the Association.

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# The Journal of the Maine Medical Association

Volume Fifty-Five

Brunswick, Maine, October, 1964

No. 10

## Abdominal Catastrophes in Infants and Children: Case Reports

RICHARD B. STEPHENSON, M.D. and EVERETT A. ORBETON, M.D.

In dealing with abdominal disasters in infants and small children requiring surgical intervention, we have repeatedly been impressed by two considerations: first, that it is frequently difficult or impossible to establish a definitive diagnosis prior to surgery, and second, that once it has been established beyond any reasonable doubt that the child has some sort of acute abdominal catastrophe requiring surgical intervention, valuable time may be lost in pursuing fruitless laboratory examinations. Meanwhile the child with peritonitis, intestinal obstruction, or devitalized bowel may rapidly become more toxic, in a greater state of fluid imbalance, and moribund while being observed and "worked-up." Also it is common for a relatively early exploration to make the difference between the necessity for performing a comparatively simple versus a very extensive and complicated operation, as well as determining the extent of morbidity.

### CASE REPORTS

A. H. This 8-month-old child was admitted to the hospital in the afternoon with a history of having been seized that morning with a sudden severe abdominal pain which caused him to scream aloud, after which he vomited. A little later he passed some bloody mucous in his stool. He was seen by his family physician, who made the diagnosis of intussusception, and sent him to the hospital for treatment. On examination he was a healthy appearing baby, crying only when his abdomen was palpated. A mass could be felt slightly to the left of the umbilicus. On combined rectal and abdominal examination it was possible to delineate the mass somewhat better, and there was bloody mucous on the examining finger. The child was immediately explored through a low right paramedian incision. The ileocolic intussusception was a little to the left of the mid-colon; this was gently reduced manually and the terminal ileum and cecum then drawn out of the wound,

covered with warm moist saline sponges and observed for several minutes. Since there appeared to be no portion of the bowel in which viability was compromised, it was gently replaced in the abdomen and the operation terminated. The child made an entirely uneventful recovery and was discharged on the sixth postoperative day.

L. G. This 9-month-old male infant was admitted through the accident division in critical condition, nearly moribund. His abdomen was enormously distended and a flat plate revealed obvious complete small bowel obstruction with fluid levels. The mother was not a very reliable historian, but the baby had been ill for several days, had had no bowel movements for at least three days and had vomited everything taken by mouth for at least three days.

Examination revealed a pale, listless dehydrated child with an enormously distended abdomen who responded only to painful stimuli. The pulse was around 200 and respirations were above 60 and shallow. A Levin tube was passed and a curdown performed. Following these procedures, the child's condition improved slightly, and an abdominal exploration was carried out as soon as possible. A complex double ileoilealileocolic intussusception was found, of which it was possible to reduce the ileocolic portion, but the ileoileal portion contained gangrenous ileum which it was necessary to resect. Since the appendix also appeared of dubious viability, it was removed. Enormous mesenteric lymph nodes were noted, and one of these removed. Microscopic examination of this showed only inflammatory infiltrate. During the immediate postoperative period the child's condition remained critical, with markedly elevated temperature, pulse and respirations, which however, gradually improved; by the sixth postoperative day peristalsis had resumed and the baby was able to take small amounts of glucose and water by mouth. After this the child improved slowly and was eventually discharged home completely recovered on the eighteenth postoperative day.

In the first of these two cases the diagnosis was obvious, and the child might have been satisfactorily treated with a diagnostic and therapeutic barium enema.

The second case was nearly moribund when first seen and was probably too ill to have survived long enough for a barium



enema, which would have been fruitless in any case, because of the gangrenous bowel involved. A definitive preoperative diagnosis was not made in this case, although intussusception was, of course, considered as a possible cause of the obvious intestinal obstruction.

E. H. This 3-month-old male infant was admitted to the hospital with no specific complaints other than listlessness and having had several loose stools on the morning of admission. The family physician found nothing specific wrong with the child but felt that he looked ill and should be hospitalized and under observation. The pediatric resident did not feel that the baby had much wrong with him. At the time of admission the attending pediatrician however, felt that he presented a diagnostic problem, as a pale, anxious appearing infant who seemed obviously ill, although without specific findings or complaints. On the following morning he had begun to vomit bile stained material; the abdomen appeared slightly distended and no peristalsis could be heard. He had had no stools since admission and would not feed. Respirations were shallow and rapid; electrolyte and other blood studies were not helpful; x-rays of the chest were negative but a preliminary flat plate of the abdomen suggested the possibility of intussusception. Barium enema however demonstrated no evidence of intussusception. The radiologist's impression was that the child had some sort of an inflammatory process in the right lower quadrant, possibly a perforated appendix. At this time the child was first seen in surgical consultation. He had a tense silent abdomen, temperature was a sub-normal 96, respirations about 50 and the pulse about 160. The child appeared virtually dead and as quickly as possible an abdominal exploration was carried out.

A long segment of the mid-small bowel was involved in a complicated volvulus and was completely gangrenous. This did not represent a classical situs inversus; the large bowel was in normal position and there were no duodenal bands. The gangrenous small bowel was exteriorized, resected and disentangled, and an end to end anastomosis performed.

The condition of the baby began to improve as soon as he had been separated from the dead bowel, and continued to do so. It was necessary to keep him on gastric suction and intravenous fluids for two days, but he steadily improved and was discharged home on the 13th postoperative day.

S. N. This one-week-old male infant was admitted to the hospital because of lethargy and failure to feed. He was the third born of the mother, and the delivery was uncomplicated and spontaneous. There are some interesting psychogenic implications in the case which it is unfortunately impossible to completely document. The baby had been a poor feeder since the time of birth and had been lethargic; slight jaundice was noted on the fifth day of life. There had been no vomiting and meconium had been passed.

On the day following readmission, intake of food was still inadequate and intravenous feedings were started. Two days later, it was noted for the first time that there was some abdominal distention and it was also felt that some edematous fluid was present within the abdominal wall. On the following morning, abdominal distention was more obvious and the baby vomited dark brown, guaiac positive material. At this time he was seen in surgical consultation, the stomach was aspirated, a cutdown performed, and an abdominal exploration carried out with the preoperative impression that he had some sort of an abdominal catastrophe or abnormality involving gangrenous bowel. On exploration, there appeared to be impaired blood supply to the entire bowel, both large and small. The colon especially was markedly edematous and did not appear viable. A cecostomy was performed, but it was our feeling that this baby was in a near terminal condition. Postoperatively, he continued to be very ill, and expired on the following day.

On post-mortem examination, no clots were demonstrated within the vascular structure of the mesentery. The small intestine showed marked congestion of the serosal surfaces, but there were no ulcers of the mucosa. The sections of the large

intestine including the cecum, ascending and transverse colon showed multiple ulcerations and marked chronic inflammatory reaction quite characteristic of ulcerative colitis.

A review of the literature since 1959 yielded only two other cases of ulcerative colitis in infants, both reported by de Boissiere, Beckeres, and Bailly in the *J. Med. Bordeaux* (131 (7) 862, 1962). Both were reported to have had the severe vascular congestion of the entire bowel which we observed in this child.

Baby Girl R. This first born female child, of 6½ to 7 months gestation, was a breech delivery in the Emergency Ward under non sterile conditions. The mother had been bleeding for two days prior to admission and also had a laparotomy with removal of a large ovarian cyst at 4½ months. The baby was without spontaneous cry but responded in 4 or 5 minutes to chest massage and was pink and crying by the time of 5 minutes of life. She was slightly cyanotic out of oxygen however and was maintained in an incubator. The birth weight was 2 lbs. 8 ozs.

Physical examination showed minimal cyanosis and obvious prematurity. She was started on gavage feedings on the third day of life, but on the fourth day it was noted that the abdomen was markedly distended and tympanitic. X-rays revealed a large amount of air under both diaphragms. At this time, a surgical consultation was obtained, 70cc. of free air aspirated from the peritoneal cavity and a tentative diagnosis of ruptured stomach made. Intracardiac adrenalin was required to establish a heart rate of only 8 to 10 beats per minute and exploration of the abdomen was carried out under local anesthesia. This revealed an intact stomach, but extensive peritonitis and multiple perforations of the near terminal ileum along the anti-mesenteric border. This segment of ileum was resected and an end to end anastomosis performed. During exploration, it was also noted she had probably bilateral polycystic kidneys. The child lived for four days following surgery. When she expired a post-mortem examination revealed very little not already noted at surgery except for a dilated left ureter associated with the polycystic left kidney. The segment of bowel resected at the time of surgery showed extensive necrosis with inflammatory reaction in its wall, highly vascularized, with foci of hemorrhage especially within the mucosa along the anti-mesenteric border. The etiology for this limited area of involvement was not determined.

Of these three cases, one, although in extremis and detected late, presented a remediable situation and did well following detachment from his gangrenous bowel. The case of ulcerative colitis, in the first week of life, probably presented a hopeless prognosis from the beginning and was certainly too ill to attempt a total colectomy at the time of surgery. The premature girl presented a remediable bowel condition, and her demise was more likely the result of her immaturity and kidney difficulties than her bowel perforations which had been remedied. Their etiology remains in doubt.

D. M. This two-year-old female infant was admitted to the hospital because of anorexia, slight nausea, fever and headache, and intermittent lower abdominal pain.

Physical examination revealed an inflamed throat and hypertrophied tonsils, the neck was supple without spasm and the abdomen was described as showing some voluntary spasm with hypo-active bowel sounds. It was felt that she had some slight right CVA tenderness. Diagnoses considered at the time of admission were meningitis, poliomyelitis, upper respiratory infection and a possible question of pyelonephritis.

A lumbar puncture was negative. The white blood count was 18,000. At this time, she was seen by the attending pediatrician, who noted that the abdomen seemed rigid, with involuntary spasm, quite severe pain on deep palpation and who requested a surgical consultation. The patient was an intelligent, cooperative little girl, lying on her right side with her knees drawn up, more comfortable in this position than in any other. She had generalized involuntary abdominal spasm, almost rigidity, with



marked rebound greatest in the right lower quadrant. The clinical impression was that she had a ruptured appendix. She was almost immediately taken to the operating room and explored with findings of a ruptured appendix; this was removed and the abdomen closed without drainage. Peritoneal culture revealed *E coli*; a throat culture done prior to surgery revealed hemolytic staph aureus. She was greatly improved on the day following surgery, made a completely uneventful recovery, and was discharged home on the 7th postoperative day.

**B. B.** This 6-year-old male infant was admitted with complaints of painful urination and defecation, and some associated diarrhea. He had apparently been well until four days prior to admission when he vomited a small amount and began to pass loose yellow stools associated with marked tenesmus, and also complained of anorexia. On the day of admission, he developed in addition, dysuria, frequency and a temperature of 103.6. The admitting impression was enteritis with a question of urological involvement, because of tenderness over the bladder, and dysuria. The bladder appeared to be distended both by percussion and x-ray, but the urine showed only a few white cells. His symptoms seemed somewhat diminished on the day following admission, but by the next day it was obvious that he was getting no better. He vomited three times and abdominal examination seemed to demonstrate some localization of the tenderness toward the right lower quadrant. It was felt that he had developed some sort of an appendiceal abscess and should be explored. This was carried out and a large mid-line appendiceal abscess was discovered. This was drained and an appendectomy was performed. He was maintained on gastric suction and intravenous fluids for four days until peristalsis returned, it became possible to remove the

Levin tube, and the drainage gradually subsided. He was discharged home on the 21st postoperative day.

Although, the mortality rate for appendicitis in recent years has reached a satisfactorily low level, these two cases illustrate that this diagnosis must not be forgotten even in the case of a very young child, and that even in small children rupture with abscess formation is possible and when this occurs it greatly increases the morbidity of the disease.

#### DISCUSSION

Surveying this limited but varied group of abdominal conditions in small children, requiring surgical intervention, it seems to us that a few general conclusions which can be made. One, we would again emphasize that the lethargic, listless infant or child with even a minimum of abdominal signs must be watched with a high index of suspicion by the pediatrician, surgeon and radiologist. No amount of laboratory information will replace close, even hourly, clinical observation of such patients.

Once the necessity of surgical intervention has been reasonably established, further delay beyond that required for decompression and a beginning at hydration will often not be tolerated by the young child, since death may be only a matter of hours away.

Dr. Stephenson, NIAMD Extramural Program, National Institutes of Health, Bethesda 14, Maryland  
Dr. Orbeton, 131 Chadwick Street, Portland, Maine

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#### ONE OF MEDICINE'S "GREATEST CHALLENGES"

Money is needed to make more facilities for (drug) research available, to provide fellowships for the extra years of training required, to enable faculty members to devote a larger portion of their time to training programs, and to stimulate interest in the field; tolerance is needed so that walls that have been built around imaginary empires can be breached, so that investigators engaged in so-called basic research will stop looking down their noses at the study of drugs, so that we can all endure through the months ahead when we are called upon to adhere to standards we are unprepared to meet; enthusiasm is needed, stemming from the knowledge that through the study of drugs, medicine can be changed even more in the next 50 years than it has in the past 50. With this kind of cooperation, we should be able to meet one of the greatest challenges that has confronted the medical profession in the past century. — Harry F. Dowling, M.D., in *J.A.M.A.*, 187:3 (Jan. 18) 1964.

# Reduction of Intraocular Pressure by Means of Osmotic Agents

KEVIN HILL, M.D.

Reduction of intraocular pressure is desirable in many ophthalmic clinical conditions. In acute angle-closure, the eye cannot long withstand the high intraocular pressure and unless this pressure is lowered quickly, irrevocable loss of vision may occur. Miotics and carbonic anhydrase inhibitors are frequently not effective but intravenous osmotic agents are almost invariably successful in promptly lowering intraocular pressure. Malignant glaucoma, a rare but serious condition which may follow glaucoma surgery, and other types of secondary glaucoma intractable to more conventional medical management may be beneficially affected by osmotic agents.

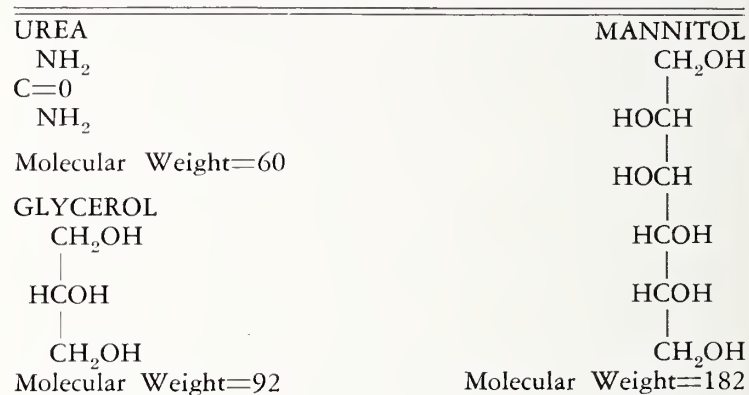
Since these agents lower intraocular pressure by drawing water from the eye into the hypertonic blood stream, the ocular contents can be relatively dehydrated. This effect is apparent in the vitreous humor and provides the rationale for the use of osmotic agents in patients undergoing cataract surgery in whom the loss of vitreous seems likely. The reduction of intraocular pressure and vitreous volume in such cases increases the chances of successful cataract extraction without the unfortunate complication of vitreous loss. For the same reason, osmotic substances may be helpful in retinal detachment surgery in those instances in which a relatively high indentation of the choroid and sclera may be needed to seal the retinal break (scleral buckling effect) but in which little subretinal fluid is available for drainage. Such indentation of the ocular coats without adequate softening of the eye by release of subretinal fluid may lead to a dangerous increase in intraocular pressure. The additional hypotensive action of osmotic substances permits a high buckling effect without such a rise in pressure.

A number of osmotic agents such as saline, sucrose, sorbitol and gum acacia have been used to lower intraocular pressure but they have proven to be unsatisfactory because of either relatively low efficacy or serious side effects.<sup>1-4</sup> More recently, however, three osmotic substances have been investigated and found to be effective ocular hypotensive agents. These substances, urea, mannitol and glycerol (Fig. 1) will be discussed seriatim.

## UREA

Hertel,<sup>5</sup> fifty years ago, demonstrated experimentally the ocular hypotensive effect of hypertonic urea but little or no clinical application of this observation was made until 1956, when Javid et al.<sup>6</sup> reported its use in lowering cerebrospinal fluid pressure in neurosurgical

FIG. 1



patients. Subsequently, Galin, Aizawa and McLean<sup>7-10</sup> reported the efficacy of urea in reducing intraocular pressure in several types of glaucoma. Their findings have since been confirmed and amplified by numerous other investigators.<sup>11-15</sup>

Urea is a substance of low molecular weight which is distributed throughout the total body water. It is distributed throughout the total body water but penetrates the blood-aqueous barrier of the eye slowly so that an effective osmotic gradient can be established. For intravenous administration, a 30% solution of urea in 10% invert sugar is used. The usual dose for glaucoma cases is 1.0 - 1.5 grams per kilogram of body weight infused at a rate of approximately three milliliters per minute. Cases with normal or only moderately elevated ocular tension will require a lower dosage to achieve a satisfactory hypotensive effect.

The following case (Fig. 2) from a series previously reported by the author will serve as an illustrative example of the efficacy of intravenous hypertonic urea.

Case 8 — A 76-year-old white woman had symptoms of acute angle-closure glaucoma for 12 hours prior to her admission to the hospital. On admission, intraocular pressure in the involved eye was 50 mm. Hg, the pupillary diameter was 4 mm., and gonioscopy revealed an entirely closed chamber angle. Despite the use of miotics and acetazolamide (Diamox®), the intraocular pressure and pupillary diameter remained unchanged for the next 10 hours. Following the intravenous administration of 1.0 gm. of urea per kilogram of body weight, intraocular pressure promptly fell to 10 mm. Hg.

The maximum hypotensive effect usually occurs within an hour after completion of the urea infusion. Shortly thereafter, the intraocular pressure begins to rise, unless surgical or additional medical means are employed, and pre-infusion pressure levels are usually attained in



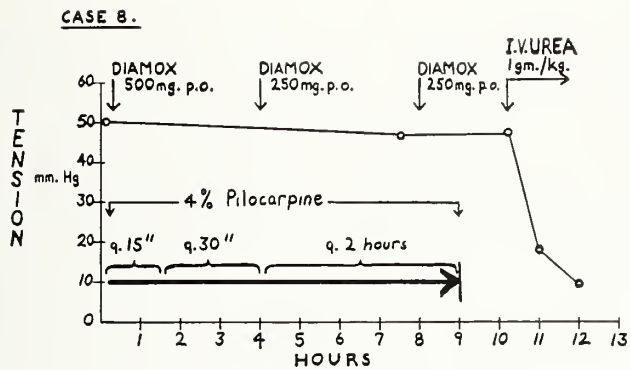


FIG. 2. Response of intraocular pressure to intravenous urea in a case which failed to respond satisfactorily to miotics (4% pilocarpine) and a carbonic anhydrase inhibitor (acetazolamide).

six to eight hours. However, the rapidity of the secondary rise in intraocular pressure may be affected by the fluid intake of the patient during and after the infusion. Thus, one patient who drank a considerable amount of water during urea administration demonstrated an unusually rapid rise in ocular tension after satisfactory hypotension had been achieved (Fig. 3).

The fall in intraocular pressure is approximately proportional to the rise in blood osmolality produced by the urea infusion. Also, a subsequent rise in tension is accompanied by a corresponding return of blood osmolality toward normal (Table I). Indeed, because of the slow rate of ocular penetration, urea may eventually exist in higher concentration within the eye than in the rest of the body fluid compartments, in which case water moves into the eye and a pressure increase above pre-infusion levels may occur. This "rebound" phenomenon has been noted and is occasionally a drawback to the use of urea.

SIDE EFFECTS

A number of side effects such as headache, thirst, dehydration, venous thrombosis and skin reaction to subcutaneous infiltration of urea solution at the infusion site have been reported.<sup>10,11,16</sup> In general, these side effects have been mild. Hemoglobinuria has been observed in monkeys receiving higher doses of urea than are customarily used in humans<sup>17</sup> but it has not been reported to occur in humans.

More serious side effects occasionally have been noted. Disorientation, mental confusion and agitation have occurred<sup>11,15</sup> and conceivably may be related to cerebral electrolyte imbalance. Intraocular hemorrhage following reduction of intraocular pressure has been observed.<sup>18,19</sup> Finally, repeated urea-induced osmotic diuresis has produced severe hyperosmolality and consequent cerebral impairment.<sup>20</sup> It should be noted that these last three side effects are most probably the result of osmotically induced changes rather than a specific noxious effect of the osmotic agent itself.

MANNITOL

Despite the obvious advantages of urea as an ocular

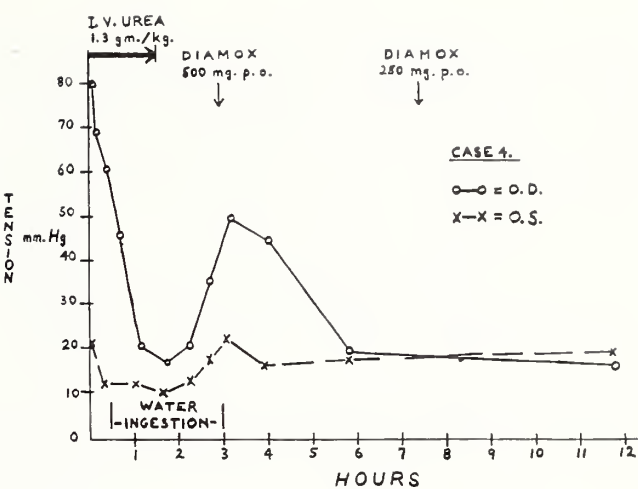


FIG. 3. Rapid secondary rise of intraocular pressure associated with excessive ingestion of water during and after intravenous urea.

TABLE I

Correlation of Changes in Intraocular Pressure with Blood Osmolality in Cases of Acute Angle-Closure Glaucoma Treated with Urea				
Case No.	Tension mm. Hg.—1948 Scale		Blood Osmolality (mOsm./L.)	
	Before Urea	After Urea	Before Urea	After Urea
2.	50	23	294	332
4.	80	18	295	315
5.	90	40	275	305
5a. <sup>1</sup>		45		295
6.	90	45	276	330
8.	46	10	282	302
10.	100	18	320	340
13.	65	14	313	380

1. As the tension began to rise in Case 5 after urea had lowered the tension to 40 mm. Hg., the blood osmolality began to fail.

hypotensive agent, the occurrence of untoward side effects prompted a search for more suitable substances. In addition, urea solutions are unstable and must be prepared by reconstitution of the lyophilized urea with invert sugar solution just prior to use. This is an endothermic process which hampers the preparation of the solution.

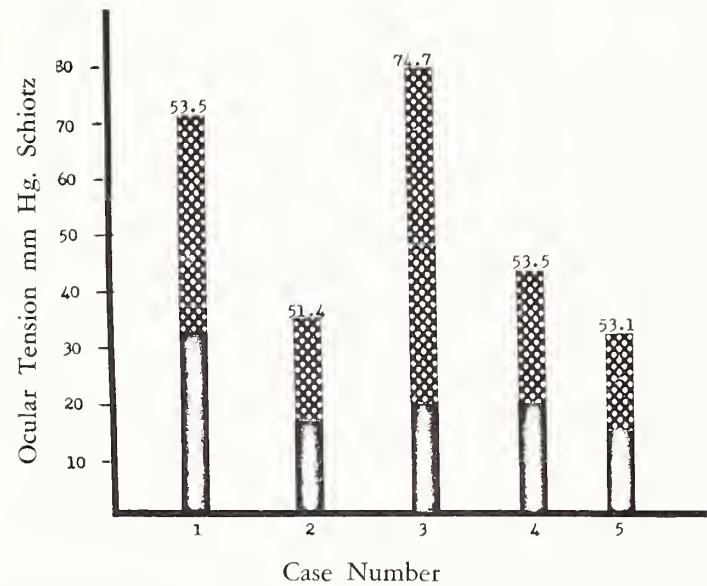
Mannitol has been found useful in other fields of medicine<sup>21,22</sup> and, more recently, has been used as an ocular hypotensive agent.<sup>23-26</sup> It is a 6-carbon hexahydric alcohol which is stable in solution and does not require mixing prior to administration. When given intravenously, it is distributed throughout the extracellular fluid compartment and is not significantly metabolized by the body. It is rapidly and totally excreted by filtration through the renal glomeruli and is not reabsorbed by the tubules.

Although mannitol has not yet been used as extensively as urea it appears to be as effective clinically. For the past two years, 20% mannitol solution has been personally administered to a series of patients with various ocular conditions (Table II, Fig. 4). In all instances, satisfactory reduction of ocular tension was accomplished.

TABLE II

Dosage of 20% Mannitol and Associated Side Effects				
Case Number	gm./kg.	Mannitol Dosage gm./kg./hr. ml./min.	Side Effects	
Angle-closure Glaucoma:				
1	1.00	1.33	9.0	None
2	1.80	1.00	5.0	None
3	3.10	2.00	10.5	Chilly Sensation
4	1.67	3.34	16.7	
5	0.66	2.64	16.7	None
6	3.00	1.50	8.3	None
Secondary Glaucoma:				
7	2.80	2.80	15.0	Transient Seizure
8	1.56	3.00	14.3	None
9	1.03	1.03	5.0	None
10	1.85	2.47	11.1	Mild Headache
11	1.80	2.40	9.0	None
"Dangerous Eyes":				
12	0.75	1.12	7.5	None
13	0.70	1.40	10.0	"
14	1.60	3.20	13.3	"
15	1.60	3.20	13.3	"
16	1.76	2.63	16.7	"
17	0.90	1.35	9.0	"
18	1.20	1.60	10.0	"
19	1.28	2.56	16.7	"
Hyphema:				
20	1.40	0.93	6.25	None

FIG. 4. The Effect of 20% Mannitol on Ocular Tension in Acute Angle-Closure Glaucoma.



Values are given for ocular tension and as a percentage fall (figures at the top of columns).

SIDE EFFECTS

In general, the side effects of intravenous mannitol appear to be the same as those encountered with urea administration. Presumably these similarities are a consequence of changes in the tonicity of the body fluids rather than the specific nature of either substance. Some differences do seem to exist, however. Mannitol is less irritating to the veins at the infusion site and does not cause the tissue damage when it infiltrates subcutaneously. Agitated confusional states have not yet been re-

ported following mannitol administration although one patient in the present series had a brief seizure characterized by sudden fleeting unresponsiveness accompanied by labored respirations and tremors of the extremities. He recovered in a few seconds and had no obvious sequellae. This episode occurred toward the end of a mannitol infusion and during gonioscopy so that the possibility of an oculocardiac reflex initiating these symptoms exists.

The rapid infusion of large doses of mannitol in patients with cardiac failure or low cardiac reserve, especially if renal function is impaired, may be hazardous due to sudden expansion of the extracellular fluid compartment and consequent overloading of the cardiovascular system. Chest pain has been noted by some patients during mannitol infusions but it is not entirely clear that these patients were experiencing angina pectoris inasmuch as some of them had normal electrocardiograms during their symptoms.<sup>25</sup>

Mannitol does not elevate the blood urea nitrogen content and thus is preferable in patients with poor renal function.<sup>27</sup> Since it is not appreciably metabolized by the body it can be safely used in patients who must restrict their carbohydrate intake.

In short, mannitol appears to be an effective ocular hypotensive agent with some minor advantages over urea in the ease of administration and as yet somewhat fewer side effects, but it has the same disadvantages inherent in the effect of all osmotic agents upon the tonicity of body fluids.

GLYCEROL

Ophthalmologists have long been familiar with the osmotic effect of glycerol when applied topically to the cornea. Cogan<sup>28</sup> and others have pointed out the usefulness of this substance in "clearing" the edematous cornea to improve visualization of intraocular structures and in the treatment of bullous keratopathy. More recently, Virno et al.<sup>29</sup> and Thomas<sup>30</sup> have reported the ocular hypotensive effect of glycerol administered orally.

Glycerol (1, 2, 3 Propanetriol) is a trihydric alcohol with a molecular weight of 92 and a specific gravity of 1.26. When given orally, it is absorbed quickly into the blood stream and metabolized in the liver. It is normally present in the body and is used both in carbohydrate and lipid synthesis. Prior to metabolism glycerol is osmotically active and relatively large amounts can be tolerated by humans without ill effect.

Virno et al. gave a 50% solution of glycerol in 0.9% saline flavored with lemon juice to their patients. They also advised chilling the mixture to improve the taste. The dosage administered ranged from 1.0 to 1.5 gm./kg. of body weight.

In a series of 26 patients with various types of glaucoma and 20 non-glaucomatous patients, they observed prompt reduction of intraocular pressure in all cases. The maximum ocular hypotension was noted in one to two hours and the hypotensive effect was maintained for three to five hours.



Thomas reported the results of orally administered glycerol in sixteen cases of glaucoma. The dosage range employed was 0.7-1.5 cc/kg. (presumably 1-2 gm/kg.) and this was administered in orange juice to make it more palatable. In all instances, the intraocular pressure fell promptly, the maximum hypotensive effect occurred in the first two hours and the effect persisted for several hours.

#### SIDE EFFECTS

The side effects noted were minimal and consisted of headache, presumably related to the concomitant reduction of cerebrospinal fluid pressure occurring in a few patients. One patient in Thomas' series reported "slight nausea" and a few other patients found the hot-sweet taste of the glycerol mixture somewhat objectionable.

Other studies<sup>31,32</sup> of the effect of orally administered glycerol to humans in doses up to 2.2 gm./kg. report a similar paucity of toxic effects. Two cases of glycerol poisoning have been reported.<sup>33</sup> One case resulted in death but the dose and route of administration were not known. The other case was that of a 2½ year old child who swallowed 300 gm. (approximately 23 gm./kg.) of glycerol but survived.

#### COMMENT

Both reports of the ocular hypotensive effect of orally administered glycerol emphasize the ease of administration and the minimal side effects. Thomas pointed out that the hazards accompanying the rapid introduction of a hypertonic solution directly into the blood stream are avoided by oral administration. Urea has also been given orally with satisfactory reduction of intraocular pressure.<sup>34</sup> The taste of urea is much more objectionable, however, and difficult to mask.

Thomas suggested that oral glycerol should be a valuable adjunct in the treatment of hyphema. It has been observed that osmotic agents given intravenously, not only lower the raised intraocular pressure which often accompanies severe anterior chamber hemorrhage but also promote the clearing of the hemorrhage.<sup>35</sup> Since hyphema frequently occurs in children in whom intravenous therapy is difficult, the availability of an effective oral osmotic agent such as glycerol should facilitate treatment of this serious ocular problem.

One case of traumatic hyphema in a young child has been treated personally and is perhaps worthy of comment.

#### CASE REPORT

A two-year-old boy\* was struck in the right eye and developed a 3 mm. hyphema. He was hospitalized but had a secondary hemorrhage which filled the anterior chamber. The ocular tension under anesthesia was 42 mm. Hg. Schiotz. A 50% solution of glycerol in isotonic saline (0.9%) was prepared and flavored with a few drops of orange extract (the patient's preference). The dose administered orally was 1 gm./kg. body

weight. In approximately three hours, the tactile tension was soft and the anterior chamber had cleared enough so that the iris periphery could be seen in two quadrants.

The boy had additional hemorrhage the following day and the ocular tension again rose. Oral glycerol was administered again with prompt reduction of tension. No side effects attributable to the glycerol were noted.

#### CONCLUSION

Ocular osmotherapy has proven to be of great value in a variety of ocular conditions marked by pathologic elevation of intraocular pressure and in other instances in which a subnormal level of ocular tension is desirable. Three osmotic agents, urea, mannitol and glycerol, are now commonly employed to achieve these ends. The present paper summarizes some of the reported and personal experience with these agents emphasizing their advantages and disadvantages. Again, it should be stressed that these agents affect the entire body and thus may produce undesirable effects upon one area or system of the body while producing beneficial effects upon the eye.

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*Continued on Page 192*

\*A patient of Dr. R. H. Dennis, to whom I am indebted for permission to report this case.

# Hospital Administration Preparation for Disaster Casualties\*

PHILIP K. REIMAN\*\*

In discussing hospital administration's preparation for disaster casualties, I am going to refer to the preparation we have made at the Maine Medical Center. However, right at the beginning, I must state that we are far from perfect in this aspect. To date, we have had only one disaster drill. Nonetheless, here are some of the things we think are right in preparing for a major disaster.

In preparing the plan, our disaster committee first secured the approval of the Medical Staff and the Board of Trustees in granting special broad powers to the Medical Disaster Chief, who is appointed by the President of the Medical Staff. This authority allows the Disaster Chief, first, the right to cancel scheduled, elective, or non-emergency operative procedures, in order to clear operating rooms for disaster victims; second, the right to directly or through designated substitutes, make available extra hospital beds, by ordering the discharge of convalescent or ambulatory patients; third, the right to order evacuation of certain parts of the hospital, by removing patients to other portions of the hospital, or to temporary quarters; fourth, the right to designate a man — or group of men — to carry on emergency and routine care of patients already in the hospital at the time of the disaster, so that the well-being of these patients will not be menaced — although their own private physician may be involved in handling disaster victims; fifth, the right to exclude, forcibly if necessary, unauthorized lay personnel from the areas being used in the treatment of disaster victims — this authority is given to both the Medical Disaster Director and the Hospital Director.

It is understood that the broad powers just named would be activated only during such periods as the disaster plan of the hospital is in action. It is assumed, of course, that the person chosen to bear the responsibility of the Medical Disaster Chief, or the Director of the Hospital would use these powers with discretion and only in the case of extreme necessity.

It must be remembered that it is essential that the need to call either an internal or external disaster program into effect must be established conclusively before such a plan is carried out. Further, it appears to us, that one of the major things to prevent is the creation of bottlenecks in the orderly flow of traffic through the disaster area.

In the case of the Maine Medical Center, it was readily agreed that the ambulance entrance and the Emergency Division would be the Triage area. In the Triage area, no definitive treatment is to be done. The major equipment to be supplied in this area besides stretchers and wheel chairs, are hemostats and tourniquets. It is the responsibility of the physicians in this area to evaluate the condition of the patients and to see that they are directed to the proper treatment areas. The disaster committee set up the following areas, all located in the immediate vicinity of the Emergency Division: first, Triage; second, Shock & Major Trauma; third, Burns; fourth, Fracture; fifth, Minor Injuries (First Aid); sixth, Major surgery; seventh, Evacuation & House Care; eighth, Consultation; ninth, Dead On Arrival; and tenth, Medical. In the Emergency Division, a large wooden box is kept, which is equipped with signs to designate each of these above mentioned treatment areas, a roll of scotch tape to affix these signs to the proper walls, as well as Patient-Identification kits, consisting of a large paper bag for patient valuables, and a Medical Record envelope, which, after being filled out, can be secured to the patient's wrist or ankle with the string provided.

Carbon copies of the medical portions of these Medical Record envelopes, concerning diagnosis, and the team to which the patient is to go, are to be filled in by the Triage officer or by a nurse, on his instructions. The Administrative records will be the Emergency Room record, completed in triplicate. Distribution is as follows: one to the Director of the Hospital, one to the Medical Disaster Chief, and one inside the Medical Record envelope attached to the patient.

The next step is to get the necessary personnel to the designated areas — this includes physicians, as well as hospital personnel. It is equally important not to get too many — nor the wrong people — at a given spot. For example, it appears to us that first-year student nurses would be of little or no value if assigned to the Triage area during the first hour of a disaster.

Proper and sufficient supplies must be readily available for the treatment areas. These supplies include medical supplies, fresh linen, stretchers, cots, blankets, and possibly, even food.

The next area that offers concern during a disaster is the control of traffic. This includes, first of all, control of traffic inside the building. For example, relatives must be directed to an area away from the immediate treatment rooms — yet, where they can receive, when available, information relating to patients about whom they

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\*\*Director, Maine Medical Center, Portland, Maine.



are concerned. It is important to keep the curious out of the treatment area, and away from the activities of the Hospital. It is also important to see that news media are kept well informed, yet, out of treatment areas. It is essential that hospital employees be directed and trained to go to areas where they can be productive, rather than becoming one of the curious — looking over the shoulders of those working in the disaster area. The control of outside traffic is equally important. It is mandatory that the Emergency entrance be kept free of parked vehicles. Therefore, provisions must be made to handle all of these conditions.

The major points of the Medical Center's Disaster Plan are: first, our method of notifying personnel within the hospital that the plan is in effect, is an announcement over the paging system, repeated many times. The phrasing used by the paging operator is: "Attention, All Personnel, Plan One Is Now In Effect." Second, depending on the time of day, outside telephone, radio, and television facilities will be used to announce that Plan One is in effect at the Medical Center. Third, employees are instructed to report directly to their team or department location. Fourth, all people are requested not to use the telephone, with the exception of Team Chiefs, who must locate their associates to notify them of the disaster. Fifth, we have requested that elevators not be used, except for official business. Sixth, we have cautioned all hospital employees to proceed with their work quietly, so that patients and visitors are not unduly alarmed. Seventh, we have directed everyone not to attempt to be an information center — but to direct newsmen to the press Information Center — and relatives, visitors, etc., to the public Information Center. Eighth, as soon as a Medical Team Chief, or a hospital department head has received notification that Plan One is in effect, it is his responsibility to notify the other members of this medical team or department, using a telephone with a 9-line, to report to the Hospital. Ninth, our disaster plan can be put into effect by the Hospital Director and the Medical Disaster Chief, or their alternates, when they receive, from a reliable source, news of a situation which could result in a large number of casualties.

Tenth, those members of the Medical Staff, not assigned by name in the Maine Medical Center plan, and those not assigned to First Aid stations by Cumberland County Civil Defense or the Mercy Hospital Disaster Plan, are to report directly to the Medical Disaster Chief, who will be located near the Triage area. These unassigned medical men are instructed not to wander around the disaster area, or join a team, without a specific assignment — as this creates confusion, and their specific skills may be badly needed elsewhere. However, the plan does provide that all medical members of anesthesia, radiological, and laboratory services are to report directly to their own departments.

Eleventh, House Staff, Residents, Interns, and medical students — many of these House Officers have specific

assignments. All unassigned members are to report to the Medical Disaster Chief or the Director of Medical Education, who will assume responsibility for the assignment of these physicians, until other attending staff members are present. If neither the Medical Disaster Chief, nor the Director of Medical Education is present, the senior Surgical Resident and the Senior Medical Resident will assume responsibility for the assignment of these personnel in the above order, until relieved by members of the Attending Staff with assigned functions.

Twelfth, the disaster plan rightfully includes the following quotation, "The Only Useful Person Is The One Who Knows His Job And Does It."

So far, we have considered the broad aspect of a hospital's preparation for disaster. Now I should like to go into some of the complex details of individual departments, whose efforts and cooperation must be utilized in order to make any mass casualty plan effective. The first department is the telephone switchboard. On Monday through Friday, from 8:00 a.m. to 5:00 p.m., they are to refer any calls, concerning a possible disaster to the Director's office. If the calling party is too upset to wait to speak to the Director, the switchboard is to obtain all possible information as to the nature and location of disaster, number of people involved, and transmit this information immediately to the Director. At all other times — nights, weekends, and holidays — the switchboard shall locate the Director or his alternate, and transmit such information as is available, concerning the extent of the Disaster. If it is decided to put the disaster plan into effect, the switchboard will announce three times over the paging system: "Attention All Personnel — Plan One Is Now In Effect." This announcement is to be repeated at regular intervals. The switchboard will also notify other key people, such as the Nursing Supervisor in charge, the senior Surgical Resident, and the Admitting Office.

At the Admitting Office, the following procedure is to be followed: on nights, weekends, and holidays, the Admitting Office secretary will be responsible for notification of personnel that cannot be handled by the switchboard. The list of these personnel is kept readily available in the Admitting office. The Admitting Director or the senior Admitting Officer present, will be assigned to coordinate the work of the Evacuation and House Care committee with the Admitting office. In no case, will the Admitting office be un-manned.

The Department of Nursing, the Director of Nursing or her alternate, upon notification that Plan One is activated, will call the senior Student Nurse resident, and request that all students report to the Nursing Assignment Center. All nursing personnel who are on duty are to remain at their stations until reassigned by competent authority. It is the responsibility of the senior Nursing Supervisor to assign sufficient personnel to the disaster teams. Head nurses assigned to in-patient areas will assume the following responsibilities: assess all personnel assigned to their nursing unit, so that those

that may be available for reassignment are known — locate and assemble all wheeled stretchers, so they may be sent to the Triage area upon request of the Medical Disaster Chief — arrange for the immediate return of all patients absent from the nursing unit. In addition, they will list on the midnight census form, the number of empty beds and the names of patients who might be subject to emergency evacuation or emergency discharge. When completed, these forms are to be sent promptly to the Evacuation and House Care Team, which will be located in the Medical Records room. Further, they are to refer all questions concerning care of patients to the Evacuation and House Care Team. They are not to attempt to page or reach individual staff physicians.

In the Emergency Division, the personnel will prepare this area for use as the Triage area, and see that all patients possible, who are not connected with a casualty, are discharged or sent to other areas of the hospital. They will arrange for an adequate supply of hemostats and tourniquets — prevent unauthorized personnel from entering the hospital, using police help if necessary — and prepare for immediate use, the supply of catastrophe records stored in the Emergency Division.

In the Housekeeping and Laundry departments, employees should report to the Housekeeping office for assignment. The Housekeeper will assign an operator to each elevator as soon as possible. She will further assign personnel to clear the coffee shop of tables and

chairs, so that it may be readied as a treatment area. Members of these two departments will stand by for messenger duty, stretcher bearers — and be available to move furniture and supplies, set up beds, and clean treatment areas. They will also clear all furniture from the classrooms to prepare them for treatment areas. Two employees will be directed to go to the Emergency Division to direct minor injury patients to the classroom area, for First Aid treatment.

Now, as you can readily see, I have mentioned only a few of the types of preparations that must be spelled out for each and every department in the hospital. And it must be noted well that the preparation of a printed plan to cover disasters is merely the first step in a major and essential project for all hospitals. It is important that once the plan has been prepared, approved by those concerned, and understood by those who must participate — regularly scheduled drills must be held, in order to disclose the weaknesses of the printed plan, as well as to familiarize all personnel with their duties. Once a disaster has occurred, it is too late for anyone to attempt to read the plan, or to understand the duties he or she is expected to perform.

The real key to the successful operation of a hospital during a disaster depends on three essentials: first, having a plan; second, knowing the plan; and third, rehearsing repeatedly, until reaction and action become automatic.

## REDUCTION OF INTRAOCULAR PRESSURE BY MEANS OF OSMOTIC AGENTS

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33 College Avenue, Waterville, Maine



# Maine Medical Association

## SPECIAL COMMITTEES 1964-1965

Special Committees for 1964-1965 as appointed by the President, Thomas A. Martin, M.D. of Portland.

### Amy W. Pinkham Fund Committee

Norman H. Nickerson, M.D., Greenville – Chairman  
Virginia C. Hamilton, M.D., South Harpswell  
Albert M. Carde, M.D., 33 Elm St., Milo  
Thomas A. Foster, M.D., 131 State St., Portland  
Ella Langer, M.D., State House, Augusta

### Diabetes Committee

Melvin Bacon, M.D., 122 Main St., Sanford – Chairman  
John S. Houlihan, M.D., 209 State St., Bangor  
Ralph Zanca, M.D., 86 Pine St., Lewiston  
Henry M. Howard, M.D., 105 Franklin St., Rumford  
Elton R. Blaisdell, M.D., 12 Deering St., Portland

### Committee On Maternal And Child Welfare

Alice A. S. Whittier, M.D., 143 Neal St., Portland – Chairman  
Robert M. Knowles, M.D., 49 Deering St., Portland  
William M. Shubert, M.D., 317 State St., Bangor  
Ella Langer, M.D., State House, Augusta  
Benjamin L. Shapero, M.D., 142 Pine St., Bangor

### Committee On Rehabilitation

John J. Lorentz, M.D., Hyde Rehabilitation Center, Bath – Chairman  
John A. Woodcock, M.D., 35 Second St., Bangor  
Edward G. Asherman, M.D., 131 Chadwick St., Portland

### Veterans Affairs Committee

William C. Burrage, M.D., 57 Deering St., Portland – Chairman  
Robert L. Ohler, M.D., Veterans Administration, Togus  
Lorrimer M. Schmidt, M.D., Veterans Administration, Togus

### Committee On Aging

James H. Bonney, M.D., 229 Vaughan St., Portland – Chairman  
Richard T. Chamberlin, M.D., 14 Gilman St., Waterville  
Philip P. Thompson, Jr., M.D., 131 Chadwick St., Portland  
Niles L. Perkins, Jr., M.D., 16 Bramhall St., Portland

### Committee On Mental Health

William E. Schumacher, M.D., 14 Westwood Rd. MD "B", Augusta – Chairman  
Francis H. Sleeper, M.D., 19 Columbia St., Augusta  
Harold A. Pooler, M.D., State Hospital, Bangor  
Margaret S. Smith, M.D., Box 967, Presque Isle  
Richard A. Levy, M.D., 22 Bramhall St., Portland  
Henry C. Thacher, M.D., 117 Goff St., Auburn

### Committee On Conservation Of Vision

Dexter J. Clough, 2nd, M.D., 224 State St., Bangor – Chairman  
Paul Maier, M.D., 723 Congress St., Portland  
Paul E. Floyd, M.D., 2 Middle St., Farmington  
Ralph A. Goodwin, Jr., M.D., 33 Court St., Auburn  
Maurice Van Lonkhuyzen, M.D., 131 State St., Portland  
Richard H. Dennis, M.D., 33 College Ave., Waterville

### Cancer Committee

Irving I. Goodof, M.D., Thayer Hospital, Waterville – Chairman  
Joseph E. Porter, M.D., 22 Bramhall St., Portland  
Charles F. Branch, M.D., Central Maine General Hospital, Lewiston

### Advisory Committee To Secretary Of State And To The Bureau Of Motor Vehicles

George L. Maltby, M.D., 31 Bramhall St., Portland – Chairman  
Milan A. Chapin, M.D., 237 Turner St., Auburn  
Wilbur B. Manter, M.D., 1 Fern St., Bangor  
Richard H. Dennis, M.D., 33 College Ave., Waterville

### Advisory Committee To Implement The Kerr-Mills Bill In The State Of Maine

Philip P. Thompson, Jr., M.D., 131 Chadwick St., Portland – Chairman  
Carl E. Richards, M.D., 34 Winter St., Sanford  
Harold N. Willard, M.D., Thayer Hospital, Waterville

### Committee To Conduct A Study Of Maternal Mortality

Robert M. Knowles, M.D., 49 Deering St., Portland – Chairman  
Eugene C. McCann, M.D., 49 Deering St., Portland  
Stanley W. Kent, M.D., 42 Deering St., Portland

### Committee To Study The Problems Of Long-Term Patient Care

Peter W. Bowman, M.D., Box C, Pownal – Chairman  
Harold N. Willard, M.D., Thayer Hospital, Waterville  
George W. Wood, III, M.D., 156 No. Main St., Brewer

### School Health Committee

Marion K. Moulton, M.D., West Newfield – Chairman  
George W. Bostwick, M.D., Newcastle  
Stanley B. Sylvester, M.D., 1377 Washington Ave., Portland  
Lloyd G. Davies, M.D., 78 Main St., Fryeburg  
Mary J. Tracy, M.D., Bristol Rd., Damariscotta

### Maine Committee — AMA-ERF

Robert W. Agan, M.D., 144 State St., Portland – Chairman  
Charles R. Glassmire, M.D., 37 Deering St., Portland  
Paul A. Fichtner, M.D., 781 High St., Bath

### \*Investment Committee

Paul S. Hill, Jr., M.D., 323 Main St., Saco – Chairman  
Adolphe J. Gingras, M.D., 105 Water St., Augusta  
Asa C. Adams, M.D., 68 Main St., Orono

\*Appointed by the Council of the M.M.A. on 8/16/64

*To be Continued*



DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## Gonorrheal Ophthalmia In A Newborn Nursery

ALTA ASHLEY, M.D., M.P.H.\*

For nearly ten years, physicians in the state have been warned in one way or another about the increasing incidence of venereal disease. Nevertheless the index of suspicion continues to be low, as shown by the following report.

On August 27, 1964 a laboratory report was received showing the presence of intracellular gram negative diplococci in pus from the eyes of a "1 week" old baby. The surname and address of this baby was noted to be the same as that of a man reported as having gonorrhea on August 20th and a woman whose disease was reported on August 24th. Immediate investigation of these cases was made by contacting the attending physician and visiting the hospital where the baby was born. The following facts were revealed.

During her pregnancy, this woman developed a vaginal discharge and at the time of her admittance in labor the discharge was profuse and foul smelling. No smears were taken.

The baby was delivered on 8/11. Immediately after birth, silver nitrate solution was instilled in the eyes and washed out with normal saline solution. She did well until 8/14 when she developed a "chemical ophthalmia" in the left eye. The following day this had spread to involve both eyes. No smears were taken and the baby was discharged on 8/15. On 8/22 the baby was seen again; the eyes were still discharging. Because the father had developed an acute gonorrheal urethritis on 8/20 and the mother still had a vaginal discharge, smears were taken on both the mother and the baby. Both showed gonococci and treatment was instigated.

There were three babies who were present in the nursery at the time the first baby was there. All of these babies left the nursery without evidence of ophthalmia.

However, their attending physicians are to contact the mothers and observe these babies for a month following discharge from the hospital.

The supply of silver nitrate used for prophylaxis was found to be discolored. On opening several perles it was found that silver deposit had formed on the walls of the wax containers. It was also found that the entire box of 100 perles had been opened and left uncovered and exposed to daylight. Incidentally, the instructions printed on the box containing the silver nitrate ampoules warned only that the drug should not be stored in a hot or cold place; did not warn against exposure to air or daylight. Deliveries in this hospital number approximately 175 a year. A box of ampoules, therefore, could be exposed to light for almost six months before all ampoules are used.

It is hoped that damage to this baby's eyes is not irreparable and that this case, which could have been prevented, will help to prevent other such incidents.

Prevention could have been carried out at several steps along the way:

1. By a diagnostic smear and adequate treatment of the mother during pregnancy.
2. By a diagnostic smear at time of delivery.
3. By proper care of and a check on deterioration of silver nitrate solution used in the delivery room.
4. By a diagnostic smear of the purulent eye discharge before baby left the hospital.
5. By raising the index of suspicion of all persons involved.

Venereal disease is a reality in our Maine population. It is time that all the diagnostic tools available are put into use as necessary, and that the possibility of venereal disease as a cause or complication must be constantly kept in mind. Blindness from gonorrheal ophthalmia must not be allowed to return.

\*District Health Officer, Augusta



## Department Plans Expanded Diabetes Detection Activities

Starting this fall, the Department — through the Division of Tuberculosis Control — plans to devote an increasing amount of time to diabetes detection. This plan has been made possible through a decrease in the 70 mm. chest x-ray programs carried on by the above division of service. According to Dr. Fisher, probably two-thirds of the field activities of the Division will now be allocated to this expanded service, the majority of which will be independent of the x-ray service.

The program will be developed on a mass community basis, limiting the service in general to all individuals thirty years of age and older. Younger individuals in any high risk groups will be included. It will be conducted initially in areas of the State where a high percentage of the residents are known to be elderly, in towns where diabetes deaths are known to have occurred year after year. A goal of 25,000 has been set for the first year of operation.

The supervisor of the Division's field activities and the two x-ray technicians are already trained in the taking of capillary bloods and the operation of the clinitron. Random sampling of capillary blood will be processed by clinitron at 160 mg.%. All bloods testing positive, at this pre-set level, will be reported to private physicians

for further patient examination and final determination of diagnosis. No re-testing will be done as part of the program.

In addition to the technical staff, staff from the Office of Health Education will assist in the necessary community organization and provision of promotional materials. Utilizing the services of the dietitian assigned to the Division for supervision of the sanatorium dietary, it is planned that she be available for consultation with physicians and, with physician approval, to develop special patient education programs concerning diabetes diets, in local areas — both on an individual and group patient basis as the situation may require.

At the present time, the Department, in conjunction with Diabetes Detection Week, 1964, is assisting with the promotion of the campaign for diabetes detection for Knox County.

It should be stated that this plan for expanded service does not in any way conflict with, nor preclude, the carrying forward of such other detection programs as may be planned in cooperation with the annual diabetes detection drive, nor those routinely established through hospitals, local health departments and the office of the private practitioner.

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### 18th Clinical Convention of the AMA

**Miami Beach, Florida — Nov. 29-Dec. 2, 1964**

A scientific program attuned to the current needs and interests of the practicing physician is planned for the 18th clinical convention of the American Medical Association.

Immunization, depression, cardiac arrhythmias, vascular occlusive diseases, emphysema, iatrogenic diseases, and hypertension are only a few of the major areas to be explored during the four-day meeting.

A new feature of the clinical convention this year is a postgraduate course of obstetrics for the general practitioner. Fifteen lectures will be presented during three sessions ranging from infertility and prenatal care through complications of labor and anesthesia to post-natal care and maternal mortality. Chairman of the course is Ralph W. Jack, M.D., Miami.

The popular fireside conferences, presented as a joint session of the American College of Chest Physicians and the AMA, will be held Sunday night, Nov. 29, at the Fontainebleau Hotel. There will be 11 tables at which 50 to 60 discussion leaders will engage in an informal and free exchange of views on a variety of medical subjects.

Six breakfast roundtables are scheduled at the di Lido Hotel. Topics include cancer of the thyroid, cosmetic surgery and peptic ulcer.

General chairmen of the local committee on arrangements are Clifford C. Snyder, M.D., and Nelson Zivitz, M.D.

## *Maine Heart Association Notes*



### **The Diagnosis of Angina Pectoris**

"The concept that the history is the essential feature in the clinical diagnosis of angina pectoris was established by William Heberden in 1768. In 1964, this is essentially unchanged, but the development of the two-step exercise test and advances in electrocardiography have given us objective studies. Recent advances in therapy make early and correct diagnosis essential so that appropriate treatment can be instituted. The diagnosis remains dependent upon the history and electrocardiographic changes, and these parameters must be critically evaluated in any patient in whom angina is considered.

"The difficulty experienced by the patient with angina is characterized as a distress or discomfort, and not a pain. The discomfort is provoked by exertion, digestion, excitement, and emotion, and is classically located in the retrosternal area, with radiation to the inner aspect of the left arm, the hypothenar eminence, and the fourth and fifth fingers. The distress may radiate to the neck, the right arm, and the jaw, and may occur in only a segment of the distribution. It develops gradually over a period of 10 seconds to 2 minutes and is relieved promptly by rest or nitroglycerin. . . .

"It is usually wise to perform a double two-step Master's test, with the test discontinued if the patient develops discomfort. Positive electrocardiographic changes after exercise include: (1) right-angle S-T segment depression of 0.5 mm. or more, (2) S-T segment elevation, (3) bundle branch block, (4) inverted T waves becoming upright, and (5) T waves increasing 5 mm. or 300 per cent in amplitude, or inverted U waves. These changes are also significant if they occur spontaneously during the period of anginal distress. . . . Negative and false-positive changes after exercise include: (1) junctional S-T segment depression of less than 2 mm., (2) T or P depression, (3) inverted T waves less than 2 mm. with or without exercise, (4) autonomic changes, and (5) false-positive results in patients with hypertrophy, bundle branch block, Wolff-Parkinson-White syndrome, after digitalization, or pericarditis."





# LOMOTIL<sup>®</sup>

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Each tablet and each 5 cc. of liquid contains:  
 diphenoxylate hydrochloride . . . . . 2.5 mg.  
 (Warning: May be habit forming)  
 atropine sulfate . . . . . 0.025 mg.

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- lowers motility promptly
- relieves spasm promptly
- stops diarrhea promptly

LOMOTIL fulfills the first order of treatment in most patients with diarrhea — prompt symptomatic control.

Pending discovery of the cause, early cessation of diarrhea is almost always urgently indicated. Prompt symptomatic control averts distress, dehydration and, frequently, severe exhaustion.

Both experimental and clinical evidence indicates that Lomotil exerts such control efficiently, safely and with maximal promptness.

#### dosage:

The recommended initial *adult* dosage is two tablets (2.5 mg. each) three or four times daily, reduced to meet the requirements of each patient as soon as the diarrhea is controlled. Maintenance dosage may be as low as two tablets daily. *Children's* daily dosage (in divided doses) varies from 3 mg. for a child of 3 to 6 months, to 10 mg. for one 8 to 12 years of age.

#### cautions and side effects:

Lomotil is an exempt narcotic; its abuse liability is low and comparable to that of codeine. Recommended dosages should not be exceeded. Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdose.

## SEARLE

*Research in the Service of Medicine*

## Necrologies

### ARMAND ALBERT, M.D.

1896-1964



Armand Albert, M.D., 67, of Owl's Head, Maine, president of the Maine Medical Association from 1956 to 1957, died suddenly on August 2, 1964 at his summer home in St. Simeon, Quebec.

He was born in St. Anne, New Brunswick on November 8, 1896, the son of Dr. Louis N. and Augustine (Hervieux) Albert.

Dr. Albert graduated from St. Mary's College in 1915 and received his medical degree from the University of Montreal in 1920. He practiced in Van Buren from 1921 until his retirement in 1963.

Dr. Albert was a member of the Maine Medical Association, the Knox County Medical Society, the American Medical Association and the Maine Chapter of the American Academy of General Practice. He was a member and a past president of the New England Council of Physicians, a member of the New

Brunswick Medical Association and at the time of his death was president of the American Cancer Society, Maine Division, Inc. Dr. Albert was also a member and past president of the Van Buren Rotary Club.

Surviving are his widow, Marie Albert of Westbrook, Maine; five sons, Louis of Montreal, Quebec; Andre of Bedford Village, New York; Marcel of East Hartford, Connecticut; Paul of Westbrook, Maine; Gilles of the Naval Air Station, Quonset Point, Rhode Island; a daughter, Mrs. Madeline Cyr of Little Nahant, Massachusetts; five brothers and 19 grandchildren.

### JAMES W. SEVER, M.D.

1878-1964

James W. Sever, M.D., 86, of Cambridge, Massachusetts died on July 19, 1964. He was born in Kingston, Massachusetts on July 4, 1878, son of Charles W. and Mary C. Sever.

Dr. Sever received his medical degree from Harvard Medical School in 1901. He interned at the Children's Hospital in Boston from 1901 to 1902 and served a residency in Surgery at the Boston City Hospital from 1903 to 1904.

Dr. Sever was an Honorary member of the Maine Medical Association and the York County Medical Society, a member of the Massachusetts Medical Society for sixty-three years, member of the American Medical Association, the New England Surgical Society, Boston Surgical Society and Société Française d'Orthopédie and a fellow of the American Orthopaedic Association, American Academy of Orthopaedic Surgeons and the American College of Surgeons.

Dr. Sever was assistant professor of Orthopedic Surgery at Harvard Medical School for many years, chief of orthopedics at Children's Hospital, Boston, associate surgeon at Boston City Hospital, orthopedic surgeon at Mount Auburn Hospital, Cambridge, visiting surgeon at York Hospital, York, Maine, and Waltham and Quincy hospitals, and medical director of the Industrial School for Crippled and Deformed Children, Boston.

Dr. Sever is survived by a daughter, two sons, four grandchildren and five stepchildren.

## County Society Notes

### SOMERSET

Fifteen members were present at a meeting of the Somerset County Medical Society at the Colony House in Lakewood, Maine on August 18, 1964. Guests included members of the Somerset County Woman's Auxiliary and Dr. and Mrs. Donald Jenkins of Webster, New York. The meeting was called to order by the President, W. Edward Jordan, Jr., M.D.

Edgar J. Smith, M.D. brought to the attention of the members that the medicare program had been renegotiated and that fees will remain about the same as they had been.

The following officers were elected for 1964-1965:

President, W. Edward Jordan, Jr., M.D., Skowhegan  
Vice-President, H. Carl Amrein, M.D., Madison  
Secretary-Treasurer, Marian L. Strickland, M.D., Canaan  
Delegates to the Maine Medical Association House of  
Delegates: George E. Sullivan, M.D., Fairfield and Har-

land G. Turner, M.D., Norridgewock. Alternates: Edgar J. Smith, M.D., Fairfield and John P. Dow, M.D., Pittsfield

Board of Councilors: Dr. Strickland; Howard L. Reed, M.D. and Maurice S. Philbrick, M.D., both of Skowhegan

Program Committee: Richard P. Laney, M.D., Skowhegan; Edgar J. Smith, M.D. and Dr. Strickland

Discipline and Ethics: William B. Grow, M.D., Fairfield; Franklin P. Ball, M.D., Bingham and Henry E. Marston, M.D., North Anson

Loring W. Pratt, M.D. of Waterville was the guest speaker. He gave a very clear and interesting presentation accompanied by colored slides covering the indications for types and care of tracheotomies.

MARIAN L. STRICKLAND, M.D.  
*Secretary*



## HANCOCK

A meeting of the Hancock County Medical Society was held at the Hancock House in Ellsworth, Maine on September 9, 1964.

Russell M. Lane, M.D. reported on the annual meeting of the Maine Medical Association House of Delegates in Rockland.

Emerson H. Drake, M.D., Chief of Surgery at the Maine Medical Center in Portland, was the guest speaker. He reported on the organization, activities and results of the Cardiac-Surgical Clinic at the Maine Medical Center and reviewed briefly the types of congenital and acquired cardiac lesions which they are correctly treating surgically together with their results.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## New Members

### CUMBERLAND

Carl A. Brinkman, M.D., 31 Bramhall St., Portland

### KENNEBEC

Richard C. Dillihunt, M.D., Maine Medical Center, Portland

## Deceased

### CUMBERLAND

Theodore C. Bramhall, M.D., 3531 Mineola Dr., Sarasota, Florida, September 3, 1964

# News, Notes and Announcements

**Department of Health and Welfare  
Division of Maternal and Child Health  
Including Services for Crippled Children  
(By Appointment Only)**

## Orthopedic Clinics

Augusta — Augusta General Hospital

1:00 p.m.: Dec. 17

Bangor — Eastern Maine General Hospital

9:00 a.m. and 1:00 p.m.: Nov. 19

*Half-day sessions 1:00 p.m.: Oct. 22, Dec. 17*

Houlton — Aroostook General Hospital

9:00 a.m.: Nov. 3

Lewiston — Central Maine General Hospital

9:00 a.m.: Oct. 16, Nov. 20, Dec. 18

Portland — Maine Medical Center

9:00 a.m.: Oct. 12, Nov. 9, Dec. 14

(In conjunction with MMC)

Presque Isle — Arthur R. Gould Memorial Hospital

9:00 a.m. and 12:30 p.m.: Nov. 4

Rockland — Knox County Hospital

1:30 p.m.: Nov. 19

Rumford — Community Hospital

1:30 p.m.: Dec. 16

Waterville — Thayer Hospital

1:30 p.m.: Oct. 22

## Cardiac Clinics

Bangor — Eastern Maine General Hospital

9:00 a.m.: Oct. 9-23, Nov. 13-20, Dec. 11-18

Portland — Maine Medical Center

9:00 a.m.: Every Friday (holidays excepted)

## COUNTY SOCIETIES

### ANDROSCOGGIN

President, Robert D. Wakefield, M.D., Lewiston

Secretary, Donald L. Anderson, M.D., Lewiston

### AROOSTOOK

President, Raymond G. Giberson, M.D., Presque Isle

Secretary, George J. Harrison, M.D., Houlton

### CUMBERLAND

President, Charles R. Geer, M.D., Portland

Secretary, Stanley B. Sylvester, M.D., Portland

### FRANKLIN

President, Stanley B. Covert, M.D., Kingfield

Secretary, Maynard B. Colley, M.D., Farmington

### HANCOCK

President, Elizabeth E. Williamson, M.D., Blue Hill

Secretary, Russell G. Williamson, M.D., Blue Hill

### KENNEBEC

President, Kenneth W. Sewall, M.D., Waterville

Secretary, Earle M. Davis, M.D., Waterville

### KNOX

President, John A. Root, M.D., Rockland

Secretary, Onni C. Kangas, M.D., Rockland

### LINCOLN-SAGadahoc

President, Edward L. Kinder, Jr., M.D., Bath

Secretary, George W. Bostwick, M.D., Newcastle

### OXFORD

President, Joelle C. Hiebert, Jr., M.D., Norway

Secretary, Albert P. Royal, Jr., M.D., Rumford

### PENOBSCOT

President, William A. Purinton, M.D., Bangor

Secretary, Hadley Parrot, M.D., Bangor

### PISCATAQUIS

President, Linus J. Stitham, M.D., Dover-Foxcroft

Secretary, Odd S. Nielsen, M.D., Dexter

### SOMERSET

President, W. Edward Jordan, Jr., M.D., Skowhegan

Secretary, Marian L. Strickland, M.D., Canaan

### WALDO

President, Norman E. Cobb, M.D., Belfast

Secretary, Seth H. Read, M.D., Belfast

### WASHINGTON

President, James C. Bates, M.D., Eastport

Secretary, Karl V. Larson, M.D., East Machias

### YORK

President, Roger J. P. Robert, M.D., Saco

Secretary, Charles W. Kinghorn, M.D., Kittery

**Cleft Palate Evaluation Clinics**

Portland — Maine Medical Center  
10:00 a.m.: Nov. 10

**Pediatric Clinics**

Bangor — Eastern Maine General Hospital  
1:30 p.m.: Oct. 23, Nov. 20, Dec. 18  
Fort Kent — Peoples Benevolent Hospital  
10:00 a.m.: Nov. 25  
Waterville — Thayer Hospital  
1:30 p.m.: Oct. 6, Nov. 3, Dec. 1

**Clinics For Mentally Retarded  
Preschool Children**

Waterville — Thayer Hospital  
9:00 a.m.: Oct. 7-21, Nov. 4-18, Dec. 2-16-30

**Adolescent Clinics**

Portland — Maine Medical Center  
1:00 p.m.: Oct. 28, Nov. 25, Dec. 23

**Cystic Fibrosis Clinics**

(In conjunction with the CMGH and MMC)

Lewiston — Central Maine General Hospital  
9:00 a.m.: Oct. 2, Nov. 6, Dec. 4  
Portland — Maine Medical Center  
9:00 a.m.: Oct. 20, Nov. 17, Dec. 15

**New England Postgraduate Assembly  
November 4, 5, & 6, 1964  
Hotel Statler, Boston, Massachusetts**

..... Lectures	..... "Gem" Lectures
..... Panels	..... Discussions
..... Symposia	..... CPC's
..... Films	..... Exhibits

Sponsored by the

Council of the New England State Medical Societies  
Registration Fee: \$15.00

**State of Maine Board of Registration of Medicine  
Secretary — George E. Sullivan, M.D.  
Waterville, Maine**

**Physicians Licensed to Practice Medicine and  
Surgery in the State of Maine  
July 7-9, 1964**

**THROUGH EXAMINATION**

Joseph B. Busheikin, M.D., 115 Torbarrie Rd., Downsview, Ontario, Can.  
Joaquim A. Cardona, M.D., 45 Rocco St., Belleville, N. Y.  
Wei-han Chou, M.D., Kingston Gen. Hosp., Kingston, Ontario, Can.  
Michael Contartese, M.D., Symmes Hospital, Arlington, Mass.  
Jacques M. Delphin, M.D., Station B, Poughkeepsie, N. Y.  
Julio Edouard, M.D., Royal Ottawa Sanatorium, Ottawa, Can.  
Feridun H. Gunduy, M.D., P.O. Box 273, E. Brentwood, N. Y.  
George Kury, M.D., 165 Longwood Ave., Brookline, Mass.  
Toh Bin Lim, M.D., 250 E. Superior St., Chicago 11, Ill.  
Eugene Martinowsky, M.D., 1601 W. Taylor St., Chicago 12, Ill.  
Pier F. Paci, M.D., 221 Longwood Ave., Boston 15, Mass.  
Alfred P. Pavet, M.D., 4840 MacArthur Blvd., Washington, D. C. 20007.

Farro Raafat, M.D., 605 W. 179th St., Manhattan, N. Y.  
Philip I. Salib, M.D., Mass. General Hospital, Boston 14, Mass.  
George Sirodot, M.D., 144 State St., Portland, Me.  
William G. Stewart, Jr., M.D., 9 Crown Ridge Rd., Wellesley, Mass.  
Ethna C. F. Winter, M.D., Willard State Hospital, Willard, N. Y.

**THROUGH RECIPROCITY**

Robert B. Allison, M.D., North Haven, Me.  
Robert P. Andrews, M.D., Falmouth, Me.  
Frank O. Avantaggio, Jr., M.D., Maine Medical Center, Portland, Me.  
Stephen Barchet, M.D., U. S. Naval Hospital, Seavey Island, Kittery, Me.  
Gerald E. Beck, M.D., 817 Med. Group, Pease AFB, N. H.  
Owen G. Bossman, M.D., U.S. NTS (T) Cutler, E. Machias, Me.  
Joseph S. Brito, M.D., Bangor, Me.  
Robert L. Callahan, M.D., Augusta, Me.  
Alroy A. L. R. Chow, M.D., Ashland, Me.  
Harry B. Eisberg, M.D., Standish, Me.  
Wesley J. English, M.D., Maine Medical Center, Portland, Me.  
Ralph J. Graff, M.D., Seal Harbor, Me.  
Lloyd E. Hawes, M.D., 59 Standish Rd., Wellesley, Mass.  
Edmund W. Hardy, M.D., Maine Medical Center, Portland, Me.  
Reginald F. Johnston, M.D., Brunswick, Me.  
David W. Khoury, M.D., 293 State St., Bangor, Me.  
Allen F. Langhorne, M.D., 202 E. New York Ave., DeLand, Fla.  
Milton F. Lehman, M.D., Surry, Me.  
James C. McLarnan, M.D., 805 E. High St., Mt. Vernon, Ohio  
Horace B. Pease, M.D., Maine Coast Regional Health Facilities, Bucksport, Me.  
Philip G. Sanfacon, M.D., Grand Isle, Me.  
Eckart Schackow, M.D., Togus, Me.  
David H. Smith, M.D., 50 Longfellow Rd., Wellesley Hills, Mass.  
Dale von Prief Fardelmann, M.D., 206 Court St., Portsmouth, N. H.  
Roland G. Ware, Jr., M.D., 16 Pinckney St., Boston 14, Mass.

**Book Reviews**

**Drugs Of Choice, 1964-1965. Edited and assembled by Walter Modell, M.D. Published by C. V. Mosby Co., 3207 Washington Blvd., St. Louis, Mo., 1018 Pp. Price \$16.75.**

This is a welcome treatise in view of the need for more unbiased information concerning our many new drugs. It is a second edition of currently used drugs, edited by Dr. Walter Modell in collaboration with forty-six other eminent authorities cogently representing various medical specialties and giving the volume a magnitude of experience in depth. It has been specifically designed as a critical review of currently used drugs. This book has nearly 900 pages packed with useful informative details of individual drug use and many helpful clinical summarization of those indicated in various clinical disease entities. Understandably, there are some drugs, of recent introduction, which are not mentioned in this text due to the necessary time lag from preparation to publication. Most of the content is of a nicely practical nature with frequent mention of hazards and side effects of drugs.

The intent of this treatise is well taken, namely, that of helping the physician "keep up to date." The latter goal has become almost impossible for the average physician in recent years because of the multiplicity of new, strange, and potent drugs and his need for time for evaluation of each.



This book should be valuable to those who read and use it in their daily practice of medicine. Critically, one wonders why whole digitalis leaf is barely mentioned or advised; and why, of the digitalis group, only the glycosides are reviewed; or, why nitroglycerine is considered next after amyl and octyl nitrites in angina pectoris, in as much as most physicians still use the former as a very reliable and practical drug.

Certainly, this book can be recommended for use by practicing physicians as a very useful source of information, aiding in the use of current drugs. It should be on each physician's desk for daily use. It is also recommended for the hospital medical library.

MILAN A. CHAPIN, M.D.  
Auburn, Maine

**Handbook Of The Practice Of Anesthesia.** By John R. S. Shields, M.B., Ch.B., F.F.A.R.C.S., Associate Professor of Anesthesiology, Department of Surgery, Washington University School of Medicine, St. Louis, Missouri. Published by C. V. Mosby Co., St. Louis, Missouri, 1963. Cloth. \$6.85. Pp 203 with 113 illustrations.

The fourteen chapters are grouped into four parts, "Pre-operative Considerations," "General Anesthesia," "Special Techniques," and "Unusual Problems Associated With the Surgery or the Patient's Condition." As a handbook it gives barely adequate step by step directions for managing various types of anesthetics and anesthesia problems, by the single method of choice based on the author's experience. Many excellent bits of advice are contained therein, and the illustrations are excellent, yet the bibliography while reasonably up to date, seems a bit short for such an all encompassing subject.

However, one is left with the feeling that, like many an international cookbook, not all countries have been best represented by the recipe selected, and on occasion some essential ingredients would appear to have been omitted. The book might well be of value to anesthesia residents or the general practitioner starting part time anesthesia, but offers little new or different to the experienced anesthesiologist.

HOWARD P. SAWYER, JR. M.D.  
Portland, Maine

**Pediatric Therapy.** Edited by Harry C. Shirkey, M.D., 1,144 pp, Published by C. V. Mosby Company, St. Louis

"Pediatric Therapy" is the result of Editor Doctor Harry Shirkey's feeling that the broad and ever-changing area of pediatric medical therapy demands a reference text. Its contributors, most well known, and all recognized experts in their respective fields have written individual sections setting forth their own therapeutic regimens. However, most sections also are notably complete, including other recognized approaches to the problem even if the individual author might not wholly agree with it.

There are those who might argue on theoretical grounds about the real need for books of this type, but the authors clearly intended to produce a reference guide for the practitioner-physician trying to keep up with advances and desiring concise authoritative comments on current therapy.

The emphasis is clearly on drug therapy. Dr. Shirkey is a pediatrician and pharmacologist and much valuable and detailed information on this aspect of therapy is included in every section. He has included several chapters on his own philosophy of the most efficacious use of drugs, and phar-

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macological principles as applied to therapeutics. A particularly excellent detailed table of dosages of a myriad of drugs is included using generic and trade names and printed on heavier colored paper for easy reference.

The format of the book is good and particularly suited to its use as a reference book. Included at the beginning of each section is a short introductory paragraph or two on pathophysiology and diagnosis of each particular entity. Adequate bibliographical references appear for those who wish to further investigate certain aspects of the material presented.

This book should be of great value to physicians dealing primarily in medical problems of children.

JOHN T. KENNEDY, JR., M.D.  
Brunswick, Maine

**The Pathogenesis of Leprosy.** By G. E. W. Wolstenholme and Maeve O'Connor, Ciba Foundation Study Group No. 15. Published by Little Brown and Co., Boston, 1963. \$2.95.

Interest in the recent advances in treatment and the pathogenesis of leprosy is, for obvious reasons, limited in Maine. Nevertheless, this up-to-date discussion on the causation of leprosy is most stimulating, as are so many of the Ciba Foundation Study Group books. Each short chapter starts with a presentation of some current work and is followed by a roundtable discussion. It is the latter, reported verbatim, which can turn the driest of subjects into one of lively interest. It is curious that in spite of all medical advances, the leprosy organism causing the human disease has yet to be cultured on any media, and two papers are devoted to describing attempts at growing it. One paper offers an intriguing explanation of the various forms of leprosy, namely that the Schwann cell has a

unique preference for engulfing the leprosy bacteria, but only when in the presence of neural damage. The commonest form of skin neural damage is from insect bites, and apparently the skin in the tropics is constantly showing superficial degeneration and regeneration of nerves as a result of insect stings. Another chapter discusses how the body reacts against dead leprosy organisms and presents the paradox of treating the leprosy with chemotherapy only to see a subsequent chronic manifestation of the disease due to the reaction to the dead organisms. Many of the changes seen in leprosy are similar to those seen in its cousin tuberculosis; this is thought by some workers to be due to the varying degree of immune response in the patient. Far more is contained in these short hundred pages than in so many fatter and more expensive volumes to be found beside it in the medical bookstore. It is recommended to those physicians entering the Peace Corps.

ANTHONY BETTS, M.D.  
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# The Journal of the Maine Medical Association

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No. 11

## Infectious Mononucleosis\*

DONAT P. CYR, M.D.\*\*

### DEFINITION

Infectious mononucleosis is an acute infectious disease which occurs chiefly in young adults; it is characterized by fever, sore throat, lymphadenopathy, splenomegaly and involvement of the liver. The disease is more specifically diagnosed by the appearance of atypical lymphocytes in the peripheral blood and by a positive heterophil agglutination test.

### PATHOGENESIS

The exact cause of infectious mononucleosis is not precisely known. It is generally considered to be a virus disease and the clinical and hematologic pictures strongly suggest a viral etiology, but the etiologic agent has not yet been convincingly demonstrated. The causative agent elicits a violent, though benign, proliferative reaction of the lymphoid and reticuloendothelial cells throughout the body. Virtually all organs and tissues can be affected, hence, the adjective "protean" is frequently used to describe the clinical manifestations of the disease. It is possible, however, to discern a distinctive pattern of the disease by careful assessment of the clinical, hematologic, and serologic findings.

### SYMPTOMS AND SIGNS

Infectious mononucleosis mainly affects young men and women in the late teens and early twenties; it is rare in patients under the age of 15 and after the age of 35. Although sporadic cases do occur, the higher incidence is encountered among students, interns and nurses, and members of the Armed Forces.

The onset is not unlike that of many viral diseases, the

most common often referred to as "La Grippe," "the flu," simple sore throat or "the virus." During the prodromal period, which lasts from one to four days, the patient complains of general malaise, lassitude, headache, anorexia, and sometimes nausea and vomiting, generalized aching, fever, and chilliness.

Dysphagia and sore throat are two of the commonest early complaints. The appearance of the throat may vary from mild redness to extensive membranous and ulcerative changes to extreme edema of the uvula and fauces. In the majority of cases there is diffuse congestion of the tonsils and pharynx with slight swelling of the tonsils. A patchy greyish membrane is seen in about 50% of the cases.

Lymphadenopathy almost invariably is present, if not early in the disease certainly in the latter part of the first week. The adenopathy may be generalized, including all the palpable lymph nodes, or confined to the neck, especially in the posterior cervical chains. Posterior cervical adenopathy is an important differential point in distinguishing infectious mononucleosis from the many bacterial infections of the throat in which the nodes at the angle of the jaw and anterior cervical chains are primarily involved.

The spleen and liver are involved in all cases and become palpable and tender in about 50% of the cases. Liver function is abnormal in almost all patients afflicted with infectious mononucleosis, and clinical jaundice is noted in 5% of the cases. It is now recognized that hepatic involvement is part of the disease entity and not a complication.<sup>1,2</sup>

Two other early signs — periorbital edema and palatine petechiae<sup>3</sup> — are diagnostically important. Periorbital edema<sup>4</sup> or, more specifically, edema of the upper eyelids gives the patient a froglike or bloated appearance and is observed in about one-third of the cases. Fine, petechial-like lesions at the junction of the

\*Presented at the American Academy of General Practice Meeting, December 7, 1963, Sanford, Maine.

\*\*Department of Internal Medicine, Lahey Clinic Foundation, Boston, Massachusetts.

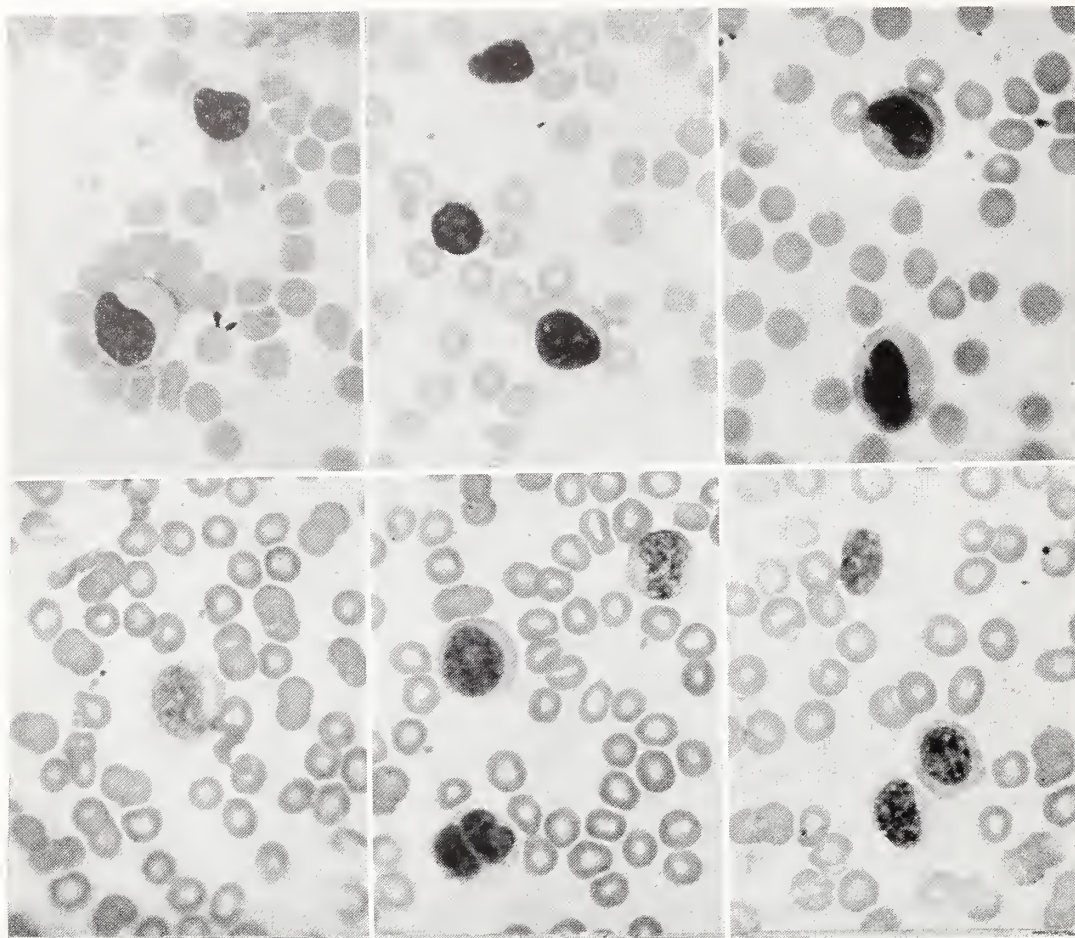


FIGURE 1.

A composite of several fields showing abnormal mononuclear cells of infectious mononucleosis. The cells in the upper fields are distinctly lymphocytoid. The cells in the lower fields have features of both lymphocytes and monocytes.

hard and soft palate have been observed early in the disease in 20 to 40% of the cases.

Although the signs and symptoms may vary in intensity and time of appearance during the first few days, the disease is usually fully developed by the end of the first week, at which time any uncertainty in the diagnosis should be dispelled by the hematologic and serologic findings.

#### HEMATOLOGY

The white blood cell count may be normal or leukopenic in the prodromal stage of the disease, but more commonly it is in the range of 12,000 to 18,000. At the height of the disease the count may be as high as 30,000 to 50,000. A lymphocytosis of 50 to 90% is usually present at some stage of the disease. Over 20% of the white blood cells are atypical lymphocytes. These have an abundant and foamy cytoplasm; they are large and mononuclear, and have some features of both lymphocytes and monocytes (Fig. 1). These atypical cells may be seen immediately on the first smear, or may not appear until the end of the first week of symptoms. The lymphocytosis usually lasts into the third week, and in most patients the differential count returns to normal between the fourth and the sixth weeks. In some patients, the atypical lymphocytes may persist for weeks or even months. These hematologic variations are illustrated in the following three cases. In the first case, early in the second week of illness, 75% of the total white blood cell count were lymphocytes, nearly 30% of which were

atypical mononuclear cells. The lymphocytosis persisted into the tenth week (Fig. 2).

In Case 2, at the end of the first week of illness, nearly 80% of the white blood cells in the peripheral blood were lymphocytes, 60% of which were large atypical mononuclear cells with foamy cytoplasm and containing vacuoles and coarse basophilic toxic granules. During the second week the lymphocytes reached a level of 90%, and in the sixth week of illness they returned to normal and only a small number still retained some toxic features (Fig. 3). In Case 3 the lymphocytosis is charted in absolute numbers (normal absolute lymphocyte count 1500 to 3000 cells per cubic millimeter). Early in the course of the illness, the total lymphocyte count was 3000, and at the height of the disease early in the second week they reached a level of 9000, at which time over one-third (or 3,500) of the circulating lymphocytes were atypical mononuclear cells. Four weeks after onset recrudescence of the disease occurred, with extensive membrane formation in the pharynx. In the sixth week of illness, the lymphocyte count was still twice the normal count (Fig. 4).

Anemia and thrombocytopenia rarely accompany the turbulent leukocytic reaction of infectious mononucleosis. This is an important point in differentiating it from acute leukemia.

Neither the lymphocytosis nor the atypical mononuclear cells are pathognomonic of infectious mononucleosis. These abnormal cells are sometimes referred to as "viocytes" because of their appearance in the



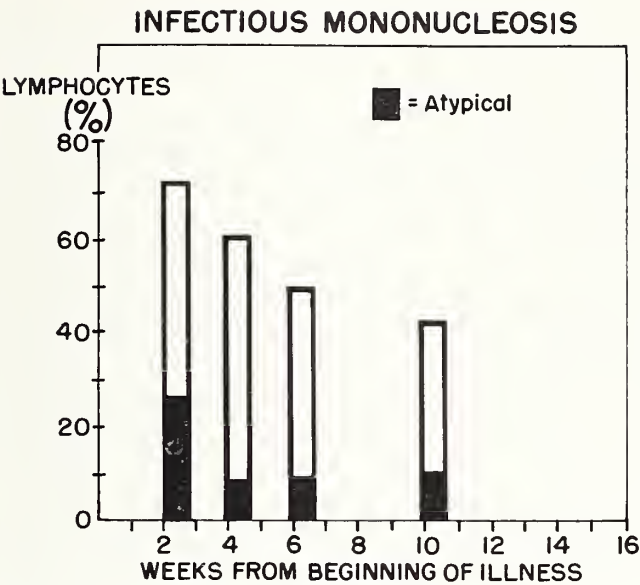


FIG. 2.

peripheral blood in many viral diseases such as infectious hepatitis, atypical viral pneumonia, measles and German measles, and even in some cases of herpes zoster. In many of these viral diseases, however, the lymphocytosis and the numbers of atypical lymphocytes rarely reach the levels found in infectious mononucleosis; more commonly, the lymphocytes are of the small mature type and are present in greatest numbers during convalescence.

SEROLOGY

At some time during the course of infectious mononucleosis, an antibody appears in the plasma which agglutinates sheep red blood cells. This is the principle which underlies the so-called heterophil (Paul-Bunnell) test. A positive heterophil test, the "typical" clinical syndrome, and the "atypical" mononuclear cells together positively identify most cases of infectious mononucleosis. The heterophil test usually becomes positive in the latter part of the first week or early in the second week of the disease (80%). In some cases, the test becomes positive in the third week or even later. Usually the test remains positive for three weeks, and in some cases the test may be positive in significant titer for many months. A titer of 1:56 is considered a positive test<sup>3</sup>; it must be recognized, however, that in such a low titer the heterophil test is merely presumptive evidence for infectious mononucleosis. The heterophil agglutinating substance may be present in healthy persons or may occur in conditions other than infectious mononucleosis: these include Hodgkin's disease, agranulocytosis, the leukemias and many other general disease states.<sup>5</sup>

A more specific test for infectious mononucleosis is the differential absorption test of Davidsohn.<sup>6</sup> The principle underlying this test is that the anti-sheep agglutinins of infectious mononucleosis are absorbed by beef red cells and are not absorbed by guinea pig kidney

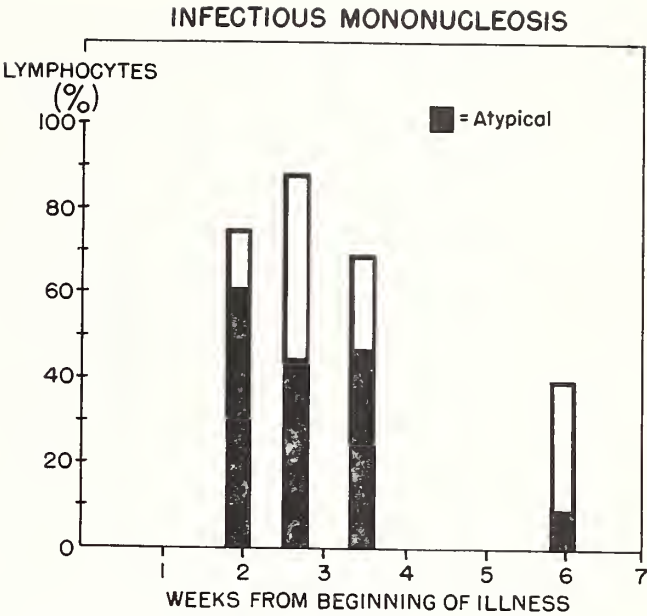


FIG. 3.

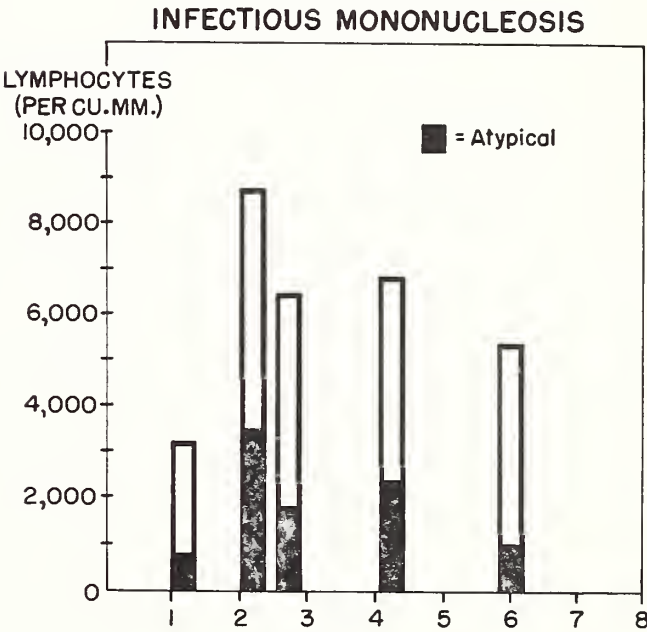


FIG. 4.

tissue. Therefore, after incubation with beef red blood cells, the serum from a patient with infectious mononucleosis will no longer agglutinate sheep red blood cells; the failure of beef cells to remove anti-sheep agglutinins is a contraindication of infectious mononucleosis. After incubation with guinea pig kidney tissue, serum from patients with infectious mononucleosis will still agglutinate sheep red blood cells in only a slightly reduced titer.

Although the differential absorption test may be of great value in the differential diagnosis of some complicated cases of infectious mononucleosis, it is not an essential test in the diagnosis of the great majority of cases, nor is it always necessary or justifiable to subject the patient to the added expense of serial heterophil agglutination tests. A simple blood smear or repeated blood smears demonstrating the large numbers of pleomorphic lymphocytes together with the typical

Table I

<i>Infectious Mononucleosis</i>	
Potentially Fatal Complications	
1. Splenic rupture	
2. Respiratory paralysis	
3. Pharyngeal complications	
a. Edema of glottis	
b. Hemorrhage from deep tonsillar ulcerations	
c. Sepsis	
4. Hematologic	
a. Hemolytic anemia	
b. Thrombocytopenic purpura	

clinical picture of sore throat and adenopathy, especially posterior cervical adenopathy in a young individual, are sufficient diagnostic criteria in most patients.

COURSE AND PROGNOSIS

In the majority of cases, infectious mononucleosis is a benign and self-limited disease. In about 70% of the cases, the disease in all its manifestations — clinical, hematologic, and serologic — completely subsides by the end of the third week of illness. The disease is even milder and of shorter duration in approximately 20% of the cases. Neurasthenic symptoms may persist for many weeks or even months in a small number of patients. This is especially true in patients with significant liver involvement, with or without clinical jaundice. Indeed, recent reports indicate that in some instances the liver involvement may slowly progress to cirrhosis.<sup>7</sup> Female patients, for some unknown reason, seem more prone to a prolonged convalescence with symptoms of apathy, difficulty in concentration, lack of interest and easy fatigability.

Recrudescence of symptoms may sometimes occur in a patient who has recovered from the acute phase and, seemingly, is well on the way to an uncomplicated convalescence. The sore throat, fever and adenopathy recur and the recurrent symptoms are sometimes worse than in the initial phase of the disease.

The question is sometimes asked: "Are there recurrences of infectious mononucleosis?" According to Bender,<sup>8</sup> genuine well authenticated cases of recurrent mononucleosis are a rarity. The same author pointed out that "more than a positive heterophil test is required to establish the diagnosis" of infectious mononucleosis. He further stated: "One never sees a patient who has clinical and serologic manifestations of infectious mononucleosis who does not also have characteristic blood cell changes." Hoagland<sup>9</sup> has recently reemphasized the phenomenon of resurgence of the heterophil-antibody reaction. A significantly elevated titer may be observed following a nonspecific upper or lower respiratory tract infection in a patient who has had infectious mononucleosis months or years previously. In summary, these

authors point out that the requirements for a diagnosis of recurrent infectious mononucleosis are the same as for the initial disease, namely, a typical clinical picture, a characteristic blood picture, and a positive heterophil agglutination test. When these criteria are applied, the instances of well authenticated cases of recurrent mononucleosis are rare indeed.

COMPLICATIONS

Although infectious mononucleosis commonly pursues a benign course, it is important to be aware of the complications, some of which may be potentially fatal (Table 1). These serious complications usually arise during the second and third weeks of the disease.

*Pharyngeal Complications and Sepsis*

The almost universal involvement of the pharyngeal lymphoid tissue in infectious mononucleosis prepares a fertile field for bacterial invaders. Culture studies and appropriate antibiotic therapy become necessary in some cases.<sup>10</sup> In rare instances pharyngeal and laryngeal edema may be so extreme as to be life threatening. With the onset of the slightest sign of laryngeal obstruction, tracheotomy must be considered. The prompt use of intravenous adrenocorticotrophic hormone (ACTH) and antibiotics has been lifesaving in this rare complication. Deaths caused by hemorrhage in the pharyngeal tissues secondary to deep ulceration have been reported.

*Central Nervous System Complications*

Pain and stiff neck are common complaints early in the course of infectious mononucleosis. These symptoms result from the posterior cervical lymphadenopathy. The same symptoms arising during the second week of disease suggest a meningitis or meningo-encephalitis if accompanied by severe headache, apathy, drowsiness and, rarely, delirium or coma. More commonly, the involvement of the central nervous system is in the nature of a peripheral neuropathy, especially affecting the cranial nerves,<sup>11</sup> or a polyneuritis, in the nature of a Guillain-Barré syndrome.<sup>11-14</sup>

In the presence of involvement of the central nervous system the spinal fluid may be normal. More commonly, however, there is an increase in cells, especially small lymphocytes, as well as an increase in pressure and in spinal fluid protein. The spinal fluid protein may be increased without increase of the cellular content, as in the Guillain-Barré syndrome. Heterophil antibodies have been found in the spinal fluid.

Although the incidence of central nervous system involvement is low (less than 1%) the mortality is relatively high (11%).<sup>15</sup> Death results from respiratory paralysis either of peripheral or central origin.

*Splenic Rupture*

The most common cause of death in infectious mononucleosis is splenic rupture. York<sup>16</sup> reviewed 45 cases reported in the literature up to January 1962. Although rare, this complication must be kept in mind; it must be recognized early in order to avoid a fatality. This complication usually arises during the second or third



week. Sudden abdominal pain, especially left upper quadrant pain referred to the left shoulder (elicited by elevating the foot of the bed) with or without shock (or tachycardia), tenderness and rigidity in the left upper quadrant, and a drop in hemoglobin are signs that should mobilize the surgical team into performing an immediate splenectomy.

Pathologic studies of the spleen have shown a massive infiltration with atypical mononuclear cells. The spleen is swollen and friable. The capsule and trabeculae become weakened by the infiltrate, and rupture or subcapsular hemorrhage may occur upon minimal straining, trauma or too vigorous palpation.

*Hematologic Complications*

Anemia and thrombocytopenia are so rare in infectious mononucleosis that their absence is an important point in differentiating this disease from acute leukemia. Both have been reported in sufficient numbers, however, to be considered important complications of this disease.

In a review of the literature up to January 1960, Green and Goldenberg<sup>17</sup> found 21 cases of hemolytic anemia complicating infectious mononucleosis. The anemia usually begins during the second or third week of the disease. It is usually slight to moderate, but it may also be severe and fulminating. The exact mechanism is not fully understood, but in some cases an auto-immune mechanism has been implicated.<sup>18</sup> Hemolytic antibodies and strongly positive Coombs' test have been demonstrated.

In most cases, the hemolytic process has been mild and self-limited. Favorable response has been noted with ACTH or cortisone.

Acute thrombocytopenic purpura, usually occurring during the second or third week, may also complicate infectious mononucleosis.<sup>19,20</sup> Grossman and Wolff<sup>21</sup> found 23 such cases in the literature up to December 1959. Again, the exact pathogenesis is not fully understood, but there is a strong probability of an auto-immune mechanism. In most cases the purpura has been self-limited and spontaneous recovery has occurred within two to six weeks. Nevertheless, it is important to recognize the potential danger of this complication, especially since a dramatic response has been noted in a few reported cases following ACTH or cortisone therapy.<sup>21</sup>

UNUSUAL PRESENTING MANIFESTATIONS AND  
DIFFERENTIAL DIAGNOSIS

The severe sore throat and the appearance of the pharynx in infectious mononucleosis may be mistaken for diphtheria or acute streptococcal sore throat (Table 2). The blood picture alone, characterized by a polymorphonuclear leukocytosis, should differentiate bacterial pharyngitis from infectious mononucleosis early in the course of the disease. Culture studies may be indicated in doubtful cases.<sup>10</sup> Because of the painful stiff neck of posterior cervical adenopathy, bacterial meningitis may be considered in the differential diagnosis. In a young

Table 2

<i>Infectious Mononucleosis</i>	
Differential Diagnosis	
1. Severe sore throat—membrane	1. Diphtheria—streptococcus pharyngitis
2. Painful stiff neck	2. Meningitis
3. Lymphadenopathy	3. Hodgkin's disease
4. Abnormal cells	4. Leukemia
5. Nodes and skin rash and sore throat	5. Secondary syphilis

Table 3

<i>Infectious Mononucleosis</i>	
Unusual Presenting Manifestations	
1. Jaundice	
2. Stiff neck; delirium; visual disturbances; coma	
3. Hematuria	
4. Hemolytic anemia	
5. Thrombocytopenic purpura	

adult the febrile illness with adenopathy may arouse suspicion of Hodgkin's disease. Leukocytosis and abnormal cells in the peripheral blood has, at times, led the unsuspecting physician to a wrong diagnosis of leukemia.

These difficulties should be easily resolved if the total picture is carefully evaluated, with special emphasis, early in the disease, on the peripheral blood picture with its pleomorphic lymphocytosis and absence of anemia or thrombocytopenia. Later, the suspicion of infectious mononucleosis is confirmed by a positive heterophil test.

Finally, in an isolated case of adenopathy, sore throat and skin rash in a young adult, the possibility of secondary syphilis must be carefully evaluated. It is important to bear in mind that the serologic tests for syphilis may be transiently positive in infectious mononucleosis. The large number of atypical lymphocytes, with a rising titer for heterophil antibodies, should differentiate these two diseases. A negative history of sexual contact and the absence of a primary lesion of syphilis may also be helpful factors in distinguishing the two diseases.

It is a well known fact (yet bears repetition) that in infectious mononucleosis there is no correlation among the severity of symptoms, the level of lymphocytosis or the level of a positive heterophil titer, and the incidence of complications. In some of the mildest cases, a physician may not be consulted until complications have developed. Some complications that have been presenting manifestations are listed in Table 3. Patients have been seen in hospital emergency rooms in a comatose state from encephalitis<sup>14</sup> complicating infectious mononucleosis.<sup>22</sup> Others have sought medical attention after the onset of a severe anemia or purpura. Only by

a careful study of such patients can the true identity of the basic disease be established.

#### TREATMENT

No specific therapy is available for infectious mononucleosis; indeed, no specific therapy is required in the majority of patients. In years past, such drugs as the arsenicals, bismuth, emetine and chloroquine have been used with indifferent results. Various antibiotics have been given with equivocal results. Many patients who have responded to antibiotics had a complicating bacterial infection. Indeed, in the occasional case in which a complicating infection is suspected, the appropriate antibiotic *must* be used in the presence of superimposed bacterial invaders detected by culture and antistreptolysin studies. All authorities are in agreement that antibiotics have no influence on the basic disease and should not be used routinely.<sup>23</sup>

Many isolated reports have appeared in the recent literature relating to the use of steroids in the treatment of infectious mononucleosis. Either ACTH or cortisone has been used successfully in the treatment of the more serious complications, such as laryngeal edema,<sup>24</sup> the Guillain-Barré syndrome, hemolytic anemia and thrombocytopenic purpura.<sup>25</sup> There is evidence, as yet inconclusive, that steroids may have a beneficial effect in shortening the course of the uncomplicated disease. It must be emphasized, however, that steroids are not necessary in the great majority of cases.

The best treatment is still the seemingly old-fashioned bed rest and symptomatic care. Patients must be kept in bed during the febrile period and physical activity restricted for one to two weeks after the fever has subsided. In the average case, the patient with infectious mononucleosis can expect a total period of incapacitation of not less than two weeks. Too early resumption of activity is likely to result in recrudescence of the disease or in a state of easy fatigability, which may last for many weeks and for which the only effective treatment is rest.

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# Lactic Dehydrogenase

ULRICH MOESER, M.D.\*

In recent years the determination of the lactic dehydrogenase activity of body fluids has received extensive investigation. The lactic dehydrogenase is an enzyme, a protein of molecular weight between 120,000 and 150,000<sup>6</sup> which catalyses the following reaction:



In our laboratory its activity is determined by measuring a colored compound formed by the pyruvic acid which remains after completion of the above reaction.

The LDH does not seem to be a single, homogeneous protein. Up to now, at least five different isozymes have been identified, which differ in electrophoretic and chromatographic properties as well as in thermal stability. The concentration of LDH in different tissues and the predomination fraction are quoted from a recent paper.<sup>8</sup>

Tissue	Total LDH conc.	Predominant isozyme
Skeletal muscle	12,400 U/mg	I (heat labile)
Heart muscle	5,620 U/mg	V (heat stable)
Brain	2,000 U/mg	V (heat stable)
Liver	1,400 U/mg	I (heat labile)
Lung	552 U/mg	II III (intermediate)
Erythrocytes	81 U/mg	V (heat stable)

From this table it can be inferred that any condition leading to necrosis would produce an elevation of LDH in the body fluids in contact with the tissues involved, and the rise would be proportional to the extent of the necrosis and to the natural content in LDH of these tissues. This assumption has been widely confirmed. So, after an infarct of the myocardium there is a marked increase in the serum LDH, which begins within 24 hours, reaches a peak between 3 to 5 days and returns gradually to normal between the 6th and the 10th day. Therefore, its rise often being earlier, reaches higher levels and maintains them longer than the SGO-T.

By measuring only the heat stable fraction (isozyme V) the interference by LDH from the liver or lungs can be eliminated, making it an even more valuable test for the diagnosis of myocardial infarctions.

Total LDH is usually elevated in chronic anemias, mainly of the hemolytic type, but is normal in blood loss anemia. It is usually high in the presence of leukemias and of advanced solid malignant neoplasms. Since the LDH content of many malignant tumors is low,

the elevation of the serum LDH in malignant disease is attributed to the associated muscular wasting. Benign neoplasms as a rule do not elevate the LDH levels.

The total serum LDH is also increased in chronic renal disease and in muscular dystrophy. It may or may not be high in acute or chronic liver disease. It is occasionally elevated in congestive heart failure and in pulmonary infarcts, but the levels tend to be considerably lower than in myocardial infarcts.

The LDH activity of effusion fluids is elevated in over 50% of the cases of malignancy involving serosal surfaces, whereas in benign conditions it has only been reported to have been abnormal in a few tuberculous effusions.

As to the levels of LDH in spinal fluid in the presence of CNS cancer, reports are as yet contradictory.

A recent discovery has been the close correlation between elevation of LDH in the urine (after removal of LDH inhibitors by dialysis) and the existence of cancer of the urinary tract.

Serum LDH has been observed to be elevated three times above normal in intestinal infarction.<sup>9</sup>

Since the erythrocytes have a much higher concentration of LDH than normal plasma, hemolysis must be avoided. The plasma or serum should be separated within 30 minutes of collection of the blood, unless the latter is stored at 4 C. At this temperature it can be stored for up to 24 hours without loss of LDH activity. Sera can be stored for one week at 0-4 C. Oxalated blood is not suitable because oxalates inhibit the LDH activity.

- Normal Value: 100-350 U/mg
- Cardiac Infarct: Elevated, fraction V (heat stable)
- Pulmonary Embolism: Elevated, fraction II, III (heat labile)
- Carcinoma, Kidney, urinary bladder: Markedly elevated urinary LDH (over 1,800 U/8 hr.)
- Intestinal Infarction: Elevated
- Hepatitis: Elevated, fraction I (heat labile)

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# Comparison of Thyroid Function Tests

ULRICH MOESER, M.D.

In recent years the diagnosis of thyroid disorders has become increasingly dependent upon the results of special tests of glandular function. The appropriate tests permit a diagnosis in milder (often subclinical) and earlier stages of thyroid disease than otherwise possible. The thyroidal function tests assay different aspects of thyroidal physiology. Of the numerous tests related directly or indirectly to thyroid function, six have been selected for comparison. They are:

1. BMR (basal metabolic rate) Normal range  $-15$  to  $+15\%$ .
2. PBI (protein-bound iodine) Normal, 4.0-8.0 microgram per 100 ml of serum.
3.  $I^{131}$  (Radioiodine uptake tests) Normal range for 24 hour epithyroidal radioactivity, 15-45%.
4.  $T_3$  (erythrocyte uptake of radioiodinated l-tri-iodothyronine) Normal range 11-19% (resin test) hyperthyroid, less than 0.86; Euthyroid, 0.86 to 1.20; Hypothyroid, greater than 1.20.
5. Photomotogram (Normal range 260-380 milliseconds) Hypothyroidism, 310 to 720, Hyperthyroidism, 160 to 310.
6. TSH (tests using thyroid-stimulating hormone).

The BMR Test correlates well with the clinical impression in hypothyroidism. It is still very valuable for following the response to therapy, including iodide, thyroid hormone, radioiodide or surgery. In these situations the PBI and radioiodine tests can be misleading.

The PBI is an expression of the iodinated serum proteins and amino acids along with trace amounts of inorganic iodine that may be loosely bound to the serum albumin fraction, with thyroxine accounting for approximately 95% of the PBI. In the Butanolextractable procedure — moniodotyrosine, diiodotyrosine, and thyroglobulin are not extracted; but, unfortunately, the commonly encountered iodinated drugs and dyes are extracted in BEI. PBI and radioiodine uptake studies generally parallel each other. They are approximately equal in accuracy. They are best employed in parallel to achieve an impression close to the degree of metabolic disorder. Drug interference will effect both tests. For example iodine preparations will depress the radioiodine uptake and increase the PBI value.

The 6 hour and 24 hour radioiodine uptake test is widely used. A low uptake does not reliably establish a diagnosis of hypothyroidism, but a normal or elevated uptake makes hypothyroidism very unlikely. Drug or dye intake, severe diarrhea, or sprue may be causes of misleadingly low radioiodine uptake without hypothyroidism.

Thyroid stimulation with TSH is an excellent test to

probe into hypothyroidism. The measurement of radioiodine uptake and PBI after injection of TSH will aid in:

- (a) determination of subclinical hypothyroidism
- (b) differentiation of primary from secondary hypothyroidism
- (c) differentiation of struma lymphomatosa from non-toxic adenomatous goiter
- (d) evaluation of need for thyroid medication in patients already receiving therapy, and
- (e) detection of remnants and metastases of thyroid carcinoma.

Radio uptake and PBI values will usually correspond and provide a rather precise counter-check.

The red cell uptake of tri-iodothyronine is primarily an indirect measure of the thyroxine-binding capacity of the serum. Elevation occurs usually in hyperthyroidism; a decrease in red cell uptake is found in pregnancy or during estrogen therapy (which elevates the thyroxine-binding proteins). The significances of the  $T_3$  test lies in the fact that the test results are not altered by iodine-containing compounds, which interfere with the PBI and the radioiodine uptake tests.

The photomotograph is a new instrument for an old clinical observation. In hypothyroidism, the reflexes are prolonged. The neural reflex arc is not altered, but muscular contraction and relaxation are influenced. It is too early to appraise the accuracy of this test. A greater usefulness will probably be found in hypothyroid states than in hyperthyroidism. A major asset of this test is that it provides an answer within a few minutes and is not affected by numerous factors which alter chemical and radioiodine tests.

## SUMMARY

The six thyroid function tests which have been compared are the most commonly employed tests. No adequately controlled study has yet been published comparing all testing methods. The ideal test method is not yet known.

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# Metabolic Features in Normocalcemic Tetany

ULRICH MOESER, M.D.

Chemical changes in the blood are among the most significant features of all forms of tetany. The changes in latent tetany differ from those in manifest tetany only in degree.

The form of tetany one commonly thinks of first, is the tetany due to lowered concentration or inactivation of a portion of the serum calcium, such as: postoperative parathyroprivie, infantile osteomalacia, maternal and neonatal tetany. Tetany due to loss or lack of absorption of calcium belongs to this group, as seen in sprue, celiac disease, and other forms of steatorrhea. All these forms of tetany have three metabolic features in common:

- (1) a low serum calcium
- (2) low urine calcium
- (3) a normal pH.

A serum calcium determination will readily confirm the diagnosis of tetany of such an etiology.

Serum calcium levels may fail to support other forms of tetany, i.e., phosphate, citrate, oxalate, and alkalotic tetany.

Clinical conditions which may lead to alkalotic tetany are:

- a) ingestion of excessive quantities of alkali (bicarbonate).
- b) prolonged hyperventilation (voluntary, hysteria, encephalitides).
- c) excessive loss of HCl (excessive gastric lavage, protracted vomiting, as in pyloric or upper intestinal obstruction).

## MAGNESIUM DEFICIENCY TETANY

As indicated in the irritability formula, the magnesium ion has essentially the same effect upon the neuromuscular irritability as the calcium ion. In experimental animals and spontaneously in cattles, low magnesium tetany can occur. Hypomagnesemia being previously reported as an etiologic factor of tetany in man, recent reports discuss the possibility that magnesium deficiency tetany may be due directly to hypomagnesemia. A second possibility of hypomagnesemia is the relative in-

## LABORATORY TESTS IN ALKALOTIC, NORMOCALCEMIC TETANY

	<i>Gastric and bicarbonate tetany</i>	<i>Hyperventilation tetany</i>
Serum calcium concentration:	normal	normal
Plasma CO <sub>2</sub> comb. power:	increased	decreased
Plasma pH:	increased	increased
Plasma Cl concentration:	decreased (gastric tetany)	
Urine:	alkalinity increased	alkalinity increased

*Phosphate, citrate and oxalate tetany* can occur by parenteral administration of these chemicals. Whereas oxalate, alkaline, or neutral sodium phosphate solutions will form insoluble calcium salts and result in a concomitant fall in serum calcium concentration, injected citrate solutions will form poorly ionizable calcium-citrate compound. These compounds produce tetany without hypocalcemia. Possibly, not only citrate but also phosphates, oxalates and bicarbonates depress the ionization of calcium.

*Alkalotic, normocalcemic tetany* can be well aligned in the following formula of neuromuscular irritability:

Irritability = 
$$\frac{(Na^{+}) + (K^{+})}{(Ca^{++}) + (Mg^{++}) + (H^{+})}$$

A decrease in the hydrogen ion (H<sup>+</sup>) concentration results in increased neuromuscular irritability with no accompanying change in the serum calcium concentration. Thus, tetany may be present in alkalosis.

activation of calcium by an excess of phosphorus which would normally have been bound by magnesium.

## SUMMARY

The metabolic changes in normocalcemic tetany are discussed and the significant laboratory tests are listed.

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# Fetal Age as Determined by X-Ray

JOHN D. SOUTHWORTH, M.D.\*

The problem of when a baby will be born has concerned physicians for years. A number of methods for determining the time of delivery have been suggested. Each has its limitations. The most reliable method is figured from the beginning of the last menstrual period. All too often this date is not known with certainty

The presence of a femoral epiphysis radiographically gives a good idea of the period of gestation for this occurs in the last four or six weeks of pregnancy. Most of us know it is not always visible even at this time. Therefore, the femoral epiphysis when seen, is of considerable significance but its absence is of little value.

It seems reasonable that if the weight of the fetus could be estimated, a fairly accurate guess at the stage of embryonal development can be made.

In the September 1961 issue of the American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, Leo Stockland and Stanton Marks in an article entitled, "A New Method of Fetal Weight Determination," presented a novel way of estimating fetal weight. Their findings were the result of studying the x-rays of 175 pregnant women. These observations show a high degree of accuracy which seem almost too good to be true. I decided to repeat their work. I examined 125 x-ray studies of expectant mothers. Ten roentgenograms were unsatisfactory, four were repeat examinations, ten were Caesarian births and one was not delivered in this hospital.

My findings completely verified the work of Stockland and Marks. As the procedures are simple, I am reporting the method in detail for interested physicians.

Plain simple anteroposterior and lateral projections of the abdomen on 14"x17" films are made. Three sets of measurements are figured. First, fronto-occipital and biparietal distances of the head are taken and added together. Secondly, fetal length is determined by measuring from the sacrum to the vault of the cranium. Thirdly, the sum of the anteroposterior and the lateral diameters of the uterus. Diagrammatically the way these measurements are made is shown in Fig. 1. Figs. 2, 3 and 4 are graphs showing the usual fetal weight for each of the above findings. The weight of the fetus as determined by each of the above methods is subject to great variation. Stockland and Marks conceived the idea of adding together the results of these three procedures. They hoped the error variations would largely neutralize one another. This they found to be true. Fig. 5 is a graph showing how to estimate fetal weight from the sum of these three combined findings. Fig. 6 shows the approximate fetal age for each fetal weight.

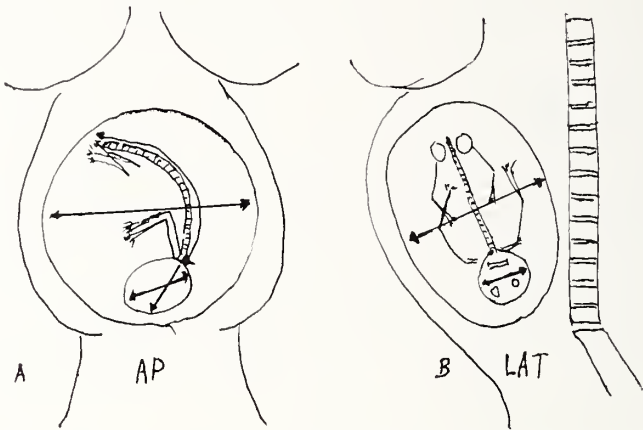


FIG. 1. (A and B) Schematic sketch showing measurements to be taken. Courtesy of Stockland and Marks.

Graph showing fetal weigh versus skull measurements.

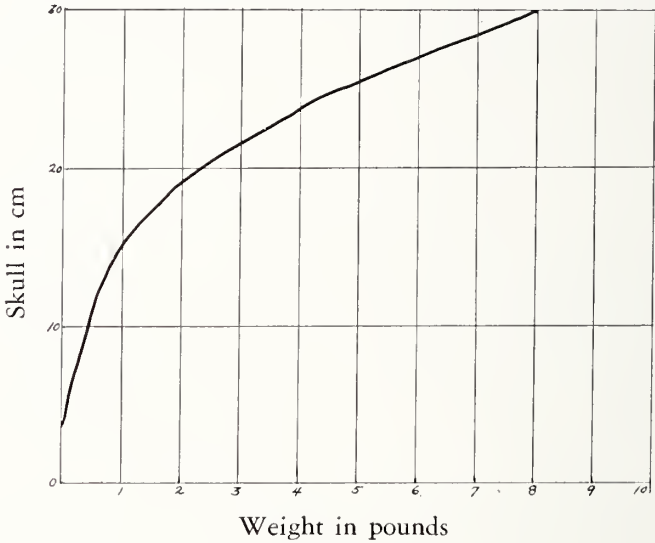


FIGURE 2

Graph showing fetal weight versus length measurements.

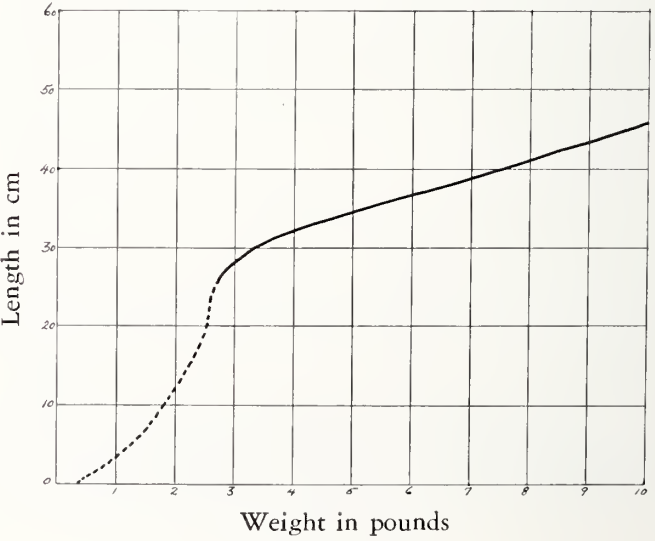


FIGURE 3

\*Scott-Webb Memorial Hospital, Hartland, Maine.



Graph showing fetal weight versus uterus measurements.

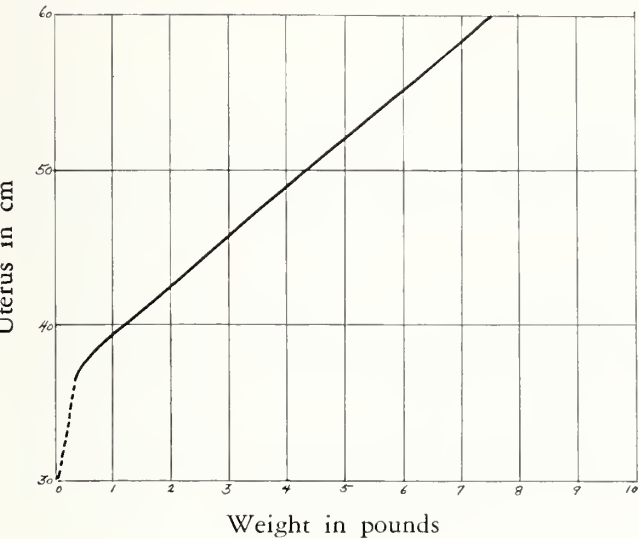


FIGURE 4

Graph showing fetal weight versus total measurements.

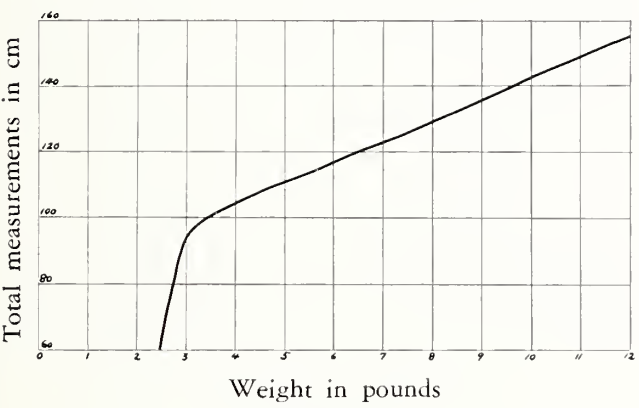


FIGURE 5

The results of our studies show that the method reported by Drs. Stockland and Marks is only slightly *less accurate* than the expectancy date figured from the patients menstrual history. It has the advantage that the estimation can be made any time after the fetus is radiographically visualized. Specifically, this is after the

Graph showing fetal weight versus weeks of gestation.

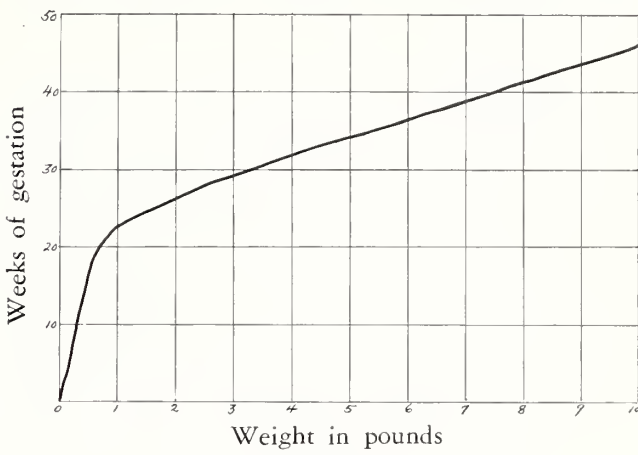


FIGURE 6

fourth or fifth month. This is not possible with femoral epiphyseal method.

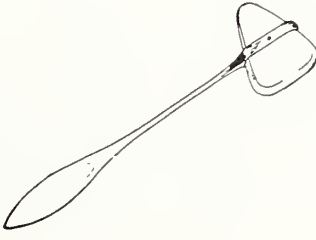
I feel that all procedures referred to above should be employed including the Stockland-Marks technique. One word of caution, the head and spine measurements are easy since the outlines are sharp but some difficulties may be encountered in determining the diameters of the uterus. The uterine shadows are more vague. However, with a little practice even these can be decided upon with moderate accuracy.

SUMMARY

1. A method of determining fetal age with a fair degree of correctness has been described.
2. The technique is relatively simple so that it is hoped doctors having fetal age problems will avail themselves of the procedure worked out by Stockland and Marks.

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Leo Stockland, M.D., and Stanton A. Marks, M.D., "A New Method Of Fetal Weight Determination." The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine. pp 425-433 September 1961.





DEAN H. FISHER, M.D.  
COMMISSIONER

## State Of Maine

# Department of Health and Welfare

## Three-Month Report on Visual Screening Amblyopia Detection Program For Three-Year-Olds in Maine

ELLA LANGER, M.D.\*

Approximately three months have elapsed since the start of the first State-wide visual screening program for all three-year-olds in Maine (reported in the May, 1964 issue of the *Journal*). The preliminary statistics are believed to hold interest for medical personnel in particular. An evaluation meeting held by the Steering Committee including allied personnel from the Department\*\* in mid-October reported early returns and findings on the program. Among other significant aspects, it was found that a surprising number of the parents' kits could not be delivered. It is believed, however, that a substantial number of these kits may have found the way eventually to the family involved as the result of repeated appeals to the target public to write in for such kits if they had not already received them.

To date, approximately one-third of the number to whom the kits were originally mailed have returned the desired information. Of these, 1.7% reported that the child tested could "see better with one eye than the other," 93% reported "no difference in the vision" and 4.4% reported that they were unable to tell whether or not the child tested "could see better in one eye than the other," or that there was "no difference." A very small percent failed to check any of the boxes indicated and so could not be counted. Twenty-nine of the children had died.

In accordance with the original plan, as fast as returns indicating need for retesting are sent in to the central office, these are referred to the proper district health office for action by the public health nurses there. These nurses, trained in the procedure, check the vision of the child on a retest chart designed for this purpose and report results immediately to the

### PROVISIONAL STATISTICS ON VISION SCREENING PROGRAM

Total Mailings		22,064	100.0
Total responses	100.0	7,004	31.7
Test result satisfactory	93.0	6,517	—
Difference between eyes	1.7	119	—
Results not clear	4.4	310	—
Results not reported	0.9	63	—
Total non-response		15,060	68.3
Returned by post office		2,000	9.1
No return		12,040	54.6

### PRELIMINARY RETEST RESULTS

Retests completed	167
"No difference between eyes"	71
"Difference between eyes"	54
Results not clear	42

central office. In several instances, volunteers within the community have been trained by public health nurse personnel to perform this function and have assisted greatly with the service.

Plans were considered at the meeting concerning follow-up on the cases referred to the physician of the family's choice and for obtaining the results of such follow-up to assure that the child receives the proper medical attention. At the same time, plans were made for a continuation of the program, as follows: that the Department carry out monthly mailings of the testing kits — possibly at the beginning of each month, starting in January of 1965 — to all parents or guardians of children of the desired age level throughout the State.

It was also agreed that the cut-off date of December 1, 1964 be set for the initial project, following which it will be written up and widely reported: that the program be tested again at a 2-year interval and comparisons made against the original report for some additional measurement of progress.

*Continued on Page XIII*

\*Director, Division of Maternal and Child Health.

\*\*Ella Langer, M.D., Chairman; John Denison, M.D., Gardiner; Richard Dennis, M.D., Waterville; Henry Thacher, M.D., Auburn; Malcolm Cass, O.D., Portland; Mary Sullivan, R.N., Edson Labrack, M.P.H., Owen Pollard, M.S., Ruth T. Clough, M.S.H. — Department personnel.



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1. Roach, T. C.: Therapy of Peptic Ulcer, J. Louisiana Med. Soc. 115:136-139 (April) 1963.
2. Steinberg, H., and Almy, T. P., Drugs for Gastrointestinal Disturbances, Chapter 21, in Modell, W. (editor): Drugs of Choice—1964-1965, St. Louis, The C. V. Mosby Company, 1964, p. 343.

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# County Society Notes

## KENNEBEC

The Kennebec County Medical Association held its first meeting of the season on September 17, 1964 in Augusta, Maine at the New Pioneer House.

The President, Kenneth W. Sewall, M.D., called the meeting to order and asked Richard H. Dennis, M.D. to give a progress report on Operation Hometown. Dr. Dennis stated that although the entire plan is now staffed and operational, it has not been activated as yet.

Paul H. Pfeiffer, M.D., in the absence of our Councilor, reported on the Maine Medical Association Council meeting held in August.

Alfred Hurwitz, M.D., surgeon at the Maine Medical Center in charge of the teaching program, was introduced by Bruce Trembly, M.D. Dr. Hurwitz spoke on the preoperative and postoperative management of surgical patients.

A meeting of the Kennebec County Medical Association was held on October 15, 1964 at the Jefferson Hotel in Waterville, Maine.

Drs. Richard H. Dennis and Eduardo A. Lopez, both practicing ophthalmology in Waterville, presented the clinical portion of the program. They illustrated their discussion of funduscopic findings in various diseases by Kodachrome projections of funduscopic examinations.

EARLE M. DAVIS, M.D.  
*Secretary*

## CUMBERLAND

A meeting of the Cumberland County Medical Society was held at the Eastland Motor Hotel in Portland, Maine on September 17, 1964 with 74 members attending.

Richard S. Hawkes, M.D. presented a resolution on the death of Theodore C. Bramhall, M.D.

Charles R. Geer, M.D. appointed the following members to serve as a Medical Advisory Committee to assist the UCS Hospital Planning and Chronic Illness Advisory Committee: Drs. Benjamin Zolov, Chairman, Emerson H. Drake, Joseph E. Porter, Charles E. Skillin, Joseph B. Earnhardt, George L. Maltby, William L. MacVane, Jr. and William J. Tetreau.

Dr. Geer also appointed Elton R. Blaisdell, M.D. as Chairman of the Diabetes Detection Week Committee with Drs. David S. Wyman and Norman W. Saunders as his committee members.

Charles R. Glassmire, M.D. reported that both of the policies approved by this society in May were acted upon favorably by the House of Delegates of the Maine Medical Association at its June meeting: the deletion of the fall clinical session and the assessment rather than contribution to the Maine Medical Education Foundation.

Niles L. Perkins, Jr., M.D. discussed the make-up, policies and problems of the State Department of Health and Welfare and its program for medical care for the aged. He stressed the need for continued medical supervision of the welfare and rehabilitation departments rather than allowing lay political control. He also emphasized the need for medical advice in health planning. Drs. Philip P. Thompson, Jr. and James H. Bonney echoed and emphasized Dr. Perkins' sentiments.

The regular meeting of the Cumberland County Medical Society was held on October 15, 1964 at the Harriet Beecher Stowe House in Brunswick, Maine with 75 members and guests attending.

Following a social hour and dinner, the Secretary read a

note from Mrs. Theodore C. Bramhall, expressing her appreciation for the resolution on the death of Dr. Bramhall which was prepared by Richard S. Hawkes, M.D. Dr. Hanley was commended for having been selected by the Maine Tuberculosis and Health Association as this year's recipient of the Huddilston Award. Kirk K. Barnes, M.D. was thanked for his assistance in arranging this meeting.

Benjamin Zolov, M.D. reported for his Medical Advisory Committee to the United Community Services, indicating that the lay members of the UCS committees had practically completed their evaluations prior to the Medical Advisory's Committee being formed. One specific recommendation by this committee was that Blue Cross-Blue Shield further increase out-patient benefits so as to lessen the burden on in-patient hospital services. After discussion by Drs. Nicholas Fish, Boris A. Vanadzin, Niles L. Perkins, Jr., Guy N. Turcotte and William L. MacVane, Jr., Dr. Philip P. Thompson, Jr. made a motion, which was passed by the membership, that a letter be sent to the UCS, recommending that the Medical Advisory Committee be a permanent liaison between the United Community Services and the Cumberland County Medical Society, with full consultation being made with this committee in all future medical matters.

Mr. Richard F. Nellson, Executive Director of the Associated Hospital Service of Maine, described the history of the Maine Blue Shield plans, the evolution of the BSA and BSC contracts from the original BSB contract, with the California relative value scale being the basis of the newer programs. He presented literature which superficially suggested that Blue Cross-Blue Shield provided more benefits per dollar than the commercial insurance plans. After a question and answer period, the meeting was adjourned.

STANLEY B. SYLVESTER, M.D.  
*Secretary*

## OXFORD

The annual meeting of the Oxford County Medical Society was held on October 7, 1964 at the Bethel Inn, Bethel, Maine. The following officers were elected for the year 1965:

President, Joelle C. Hiebert, Jr., M.D., Norway  
Vice-President, Henry M. Howard, M.D., Rumford  
Secretary-Treasurer, Ake Akerberg, M.D., South Paris  
Delegates to the Maine Medical Association House of Delegates: Peter B. Aucoin, M.D., Rumford (1 yr.) and James A. MacDougall, M.D., Rumford (2 yrs.).  
Alternates: Walter G. Dixon, M.D., Norway (1 yr.) and Albert P. Royal, Jr., M.D., Rumford (2 yrs.)

John T. Konecki, M.D. of the St. Mary's General Hospital spoke on Treatment of Mass Casualties in a General Hospital.

ALBERT P. ROYAL, JR., M.D.  
*Secretary*

## YORK

A meeting of the York County Medical Society was held on October 14, 1964 at the York Hospital in York, Maine.

The meeting was called to order by the President, Roger J. P. Robert, M.D. A nominating committee, consisting of Drs. Paul S. Hill, Jr., Carl E. Richards and Stephen A. Cobb, was appointed by the President to prepare a proposed slate of officers for 1965.

CHARLES W. KINGHORN, M.D.  
*Secretary*

*Continued on Page 218*



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1. Subacute Bacterial Endocarditis — Unusual Features
2. Bacterial Meningitides
3. Viral Infections of the Respiratory Tract
4. Viral Exanthems

### **Third Session, Saturday Morning December 5, 1964 — 9:00 A.M. to 12:30 P.M.**

1. Basic Principles for the Use of Antimicrobial Agents
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3. Present Status of Chemoprophylaxis of Infections
4. New Facts about Old Antimicrobial Agents
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COUNTY SOCIETY NOTES — *Continued from Page 217*  
HANCOCK

The Hancock County Medical Society met at the Hancock House in Ellsworth, Maine on October 14, 1964.  
Philip L. Gray, M.D. was elected chairman of the Diabetes Drive this year.  
Mason Trowbridge, Jr., M.D. spoke about Folic Acid Deficiency Anemias, their frequency, etiology and treatment.  
RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

LINCOLN-SAGADAHOC

A meeting of the Lincoln-Sagadahoc County Medical Society was held at The Ledges in Wiscasset, Maine on October 20, 1964.  
The meeting was called to order by the President, Edward L. Kinder, Jr., M.D. The Constitution and By-laws of the county society were presented for discussion and interpretation.  
The difficulty of obtaining speakers for monthly meetings of a relatively small society was discussed and a committee, consisting of the following members, appointed to oversee educational programs: Drs. John F. Andrews, Chairman, Samuel L. Belknap and Paul A. Fichtner.  
George W. Bostwick, M.D. led a discussion of intestinal obstruction with presentation of two illustrative cases of ileus.  
GEORGE W. BOSTWICK, M.D.  
*Secretary*

WASHINGTON

A regular meeting of the Washington County Medical Society was held at the Charlotte County Hospital in St. Stephen, New Brunswick, Canada on October 21, 1964.  
A. J. MacLeod, M.D., Assistant Professor of Medicine of Dalhousie University, spoke on Virus Diseases.

The following officers were elected for 1965:  
President, Robert G. MacBride, M.D., Lubec  
Vice-President, John W. McAllister, M.D., Lubec  
Secretary-Treasurer, Karl V. Larson, M.D., East Machias  
Board of Censors: George B. Shaw, M.D., Jonesport (3 yrs.)  
Delegate to the Maine Medical Association House of Delegates: Hazen C. Mitchell, M.D., Calais. Alternate: James C. Bates, M.D., Eastport  
KARL V. LARSON, M.D.  
*Secretary*

New Members

CUMBERLAND

Aldo F. Llorente, M.D., 56 Baribeau Dr., Brunswick  
Louis N. Taxiarchis, M.D., 144 State St., Portland

KENNEBEC

Robert L. Callahan, M.D., 105 Water St., Augusta  
Richard R. Dole, M.D., 218 Main St., Waterville  
L. Armand Guite, Jr., M.D., 45 Elm St., Waterville  
Joseph J. Hiebel, M.D., 34 Gilman St., Waterville

OXFORD

Manuel G. Pena, M.D., 82 Maine Ave., Rumford

YORK

Harry B. Eisberg, M.D., 331 Main St., Saco

Deceased

Wallace E. Webber, M.D., 297 Main St., Lewiston, September 15, 1964

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I certify that the statements made by me above are correct and complete.  
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## DEPARTMENT OF HEALTH &amp; WELFARE

*Continued from Page 214*

A word of praise should be included here for the splendid response to the program from all mass media sources. A vigorous publicity campaign supportive to the program was launched currently with the initial mailing, including newspaper articles; house-organ articles and notices; one-minute TV spots on all TV stations; myriad numbers of one-minute or briefer "spot" radio announcements; an explanatory letter to all PTA groups in the State through the courtesy of the Maine Congress of Parents and Teachers; a mass mailing directly to all physicians in the State explaining the program and urging their utilization of the test as routine office procedure with this age group.

Inquiries concerning the program from other states' health department personnel and interested physicians over the country have been received by the program director, as well as requests for testing materials and for a report of the subsequent progress of the program. Although similar programs are known to have been developed on a sporadic basis throughout the United States, it is believed that the Maine program is the first of its kind to undertake the testing of the pre-school child on a State-wide mass mailing basis. If the final evaluation holds true to early promise for it, this

may well become a generally accepted and readily manageable approach to the problem.

Although returns are still coming in, it is felt that some general assumptions can be drawn about the program to date. The most significant appears to be that the procedure will work; that the anticipated clinic organization for retesting will not be needed since those screened out for retesting occur in such widely scattered areas as to render the above possibility unnecessary; that the procedure will be increasingly adopted as routine practice by the family physician during the physical examination of the three-year-old child.

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# THE ARTHRITICS WHO COULD NOT TAKE STEROIDS

The bane of the steroids, new and old, has been the certain undesirable metabolic effects—including salt and water retention, edema, overstimulation of the appetite, excessive weight gain, mood swings seemed to be firmly linked to the primary anti-inflammatory action. For arthritics already overweight or with cardiovascular disease complicated by edema or those who were tense and anxious, steroid treatment could aggravate their problems. But with the advent of ARISTOCORT® Triamcinolone, many of these arthritics became “steroid-treatable.” The reason: Not only did *this* steroid provide gratifying relief of inflammation and pain, but it did so *without* the penalty of overstimulation of the appetite, excessive weight gain, salt and water retention, edema, and undesirable euphoria. Six years of widespread use have confirmed these benefits for other arthritics as well as those formerly untreatable.







# The Journal of the Maine Medical Association

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Volume Fifty-Five

Brunswick, Maine, December, 1964

No. 12

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## *Editorial . . .*

In December 1963 the precedent was established of publishing the paper chosen by the Maine Association of Medical Technologists as the best paper to be submitted in a contest among students of laboratory technology in the State of Maine. The article on "Lipids and Lipid Metabolism" by Ottina Mertz of Waterville was well received.

This year at the annual seminar of the Maine Association of Medical Technologists the first prize for papers submitted by students was awarded to Deonne (Mrs. Robert) Jackson, a student at the Eastern Maine General Hospital, for her paper on "Periarteritis Nodosa." We are again pleased to acknowledge the fine efforts of this society in their excellent programs for continuing education in this paramedical field.

The program at the annual seminar was of excellent quality and was aimed at Quality Control in the Laboratory. The morning session, devoted to quality control in chemistry, was led by Thomas Asher, Ph.D. from the Scientific Division of Hyland Laboratories in Los Angeles, California. The afternoon session devoted to quality control in hematology, was under the direction of Dennis Dorsey, M.D., Director of Laboratories of Central DuPage Hospital, in Winfield, Illinois. Dr. Dorsey is well-known to the pathologists of this country for his important contributions to the Commission on Continuing Education of the American Society of Clinical Pathologists.

We extend congratulations to the winner of the award and our congratulations to the Maine Association of Medical Technologists for their continued efforts to maintain the high standards of the practice of medical technology in this state.

R.C.W.

# Periarteritis Nodosa

DEONNE JACKSON

Periarteritis nodosa is a widespread disseminated and inflammatory disease of the arterial system, characterized by focal inflammatory lesions involving both small and medium sized arteries. It is generally included with other diseases that are centered primarily in the tissues that possess collagen, the so called "collagen diseases" such as systemic lupus erythematosus, scleroderma and dermatomyositis. Since the cardio-vascular system is rich in collagen, in periarteritis nodosa there is widespread involvement of the cardiovascular structures and associated organs. This results in inflammatory and degenerative changes and visible alterations in the involved arteries and tissues.

Numerous cases of the disease have been reported since it was first introduced and defined by Kussmaul and Maier in 1866, yet many of these cases have been reported quite recently. This greater number of reported cases has, of course, led to a greater knowledge of the disease and its manifestations.

Kussmaul and Maier described an inflammatory disease of the medium sized arteries and named it periarteritis nodosa. This and other early reports described macroscopic nodules and it was believed that the inflammation was chiefly in the adventitia and outer media, thus the term periarteritis nodosa. Years later, Eppinger observed the disease microscopically and described it much as it appears today. This shift of emphasis from the macroscopic to microscopic form of the disease led to the term polyarteritis to designate the same disease since it was then found that the inflammatory process was not confined to the outer wall of the vessels, but affected all the coats of the artery. To add to the confusion in the history of the disease, it has also been called panarteritis, essential polyangiitis, necrotizing arteritis as well as polyarteritis and the term presently used and accepted by the members of the medical profession, periarteritis nodosa.

Many theories concerning the causative agent of periarteritis nodosa have been suggested but as yet none have been satisfactorily confirmed or completely accepted.

Syphilis was first suggested as the causative agent but this was abandoned when they found that most cases occurred in the absence of syphilitic infection. Later, many other etiologic factors were suggested such as bacterial or virus infections, toxic injury, and sensitization by rheumatic fever or some other foreign antigen. The sensitizing antigen might be bacterial in nature or perhaps one of the commonly used drugs such as sulfonamides, acetate, penicillin, or any number of other drugs.

As yet, there has been no experimental or cultural

proof of infection and the progressive nature of the disease makes it seem unlikely that it is due to drug or bacterial sensitization, since the disease continues even in the absence of the suspected antigen.<sup>7</sup> However, in the 1930's, Rich and Gregory produced lesions in rabbits experimentally as a result of hypersensitivity to foreign proteins, which closely resembled lesions produced in periarteritis associated with drug exposure, especially sulfonamides.<sup>1</sup> Furthermore, there has been an increase in the number of reported cases since 1936, the year sulfonamides were introduced. However, this and even a history of drug administration in many reported cases could be coincidental.

The most recent theory is that periarteritis nodosa is an auto-immune disease—an interaction of an autoantigen and an antibody. Since a nonsensitizing substance may become antigenic when linked with a protein, it is possible that either drugs or bacteria may attach themselves to the surface of a cell and alter it in such a way that it becomes antigenic. Furthermore, the therapeutic effect of a drug may potentiate the exposure to a bacterial protein which then could cause the host's tissue to become autoantigenic.<sup>7</sup> Thus bacteria or drugs might cause autoimmune reactions in the body. Several other factors that may possibly cause autoimmune reactions have also been suggested. Genetic factors such as asthma or hay fever, or tissue injuries such as damaging diseases (hepatitis) or excessive sunlight may also alter tissues and make them more susceptible to autoimmune reactions.

There are several factors which periarteritis nodosa and autoimmune diseases have in common. In both, there is an increased plasma, globulin and an accumulation of lymphocytes and plasma cells in the tissues damaged. However in autoimmune diseases, an antibody can be readily demonstrated and since this can not yet be done in periarteritis nodosa, the question of the etiologic basis remains to be solved.

Periarteritis nodosa may occur at all ages, although the majority of the patients reported are between 20 and 40 years of age and predominantly males. It creates the impression of an infectious disease, with either a sudden or gradual onset and a course that may be acute, subacute, or chronic. The general symptoms include malaise, anorexia, weight loss, general debility, fever, and weakness as well as other symptoms referable to the various organs and tissues affected. The fever is usually low grade, and may be either continuous or intermittent.

Since the arterial system supplies all parts of the body, symptoms may indicate involvement of many



organs and thus simulate other diseases. Most authorities agree that the most commonly involved organs are the kidneys, heart, liver, gastrointestinal tract, muscles, and occasionally the lungs and central and peripheral nervous systems. However, no region of the body is exempt.

During the early course of the illness, the patient may experience attacks of severe gastrointestinal pain — usually associated with intra-abdominal vascular lesions.<sup>2</sup> These abdominal symptoms are frequently the presenting complaint and are dependent on the location and the extent of involvement of arteries supplying the abdominal viscera. They include nonspecific abdominal pain and tenderness, nausea, vomiting, anorexia, and diarrhea. Sometimes hepatomegaly and jaundice may be noted. Serious complications often ensue and include perforation of an ulcer, ulcerative colitis, and pancreatic necrosis as well as widespread infarction.

The most frequent renal manifestation is hypertension which is probably due to vascular or other changes in the kidney. It may be pre-existent and may develop without demonstrable vascular lesions. Other renal symptoms include oliguria, nephrosclerosis, and acute glomerulonephritis: a high percentage of patients die in uremia. Rarely, the only signs of renal involvement may be slight hematuria and albuminuria.

Tachycardia and systolic apical murmur are common when the coronary arteries are involved. Anginal pain is uncommon, yet myocardial infarctions and pericarditis are found frequently at necropsy with cardiac failure being a frequent cause of death.

Involvement of the pulmonary arteries is quite uncommon and usually appears when an allergic condition, such as asthma, is present. Sometimes pulmonary symptoms such as wheezing, dyspnea, and cough develop, even without histologic evidence of lesions in the lungs. When lesions are present the patient may occasionally exhibit cyanosis, pleuritis, or hemoptysis with bronchopneumonia as a terminal manifestation.

Neurological symptoms are varied and depend on the degree and the location of the vascular involvement. Usually they are due to peripheral neuritis but may occasionally include the central nervous system. The most common symptoms are motor weakness, paresthesias, muscular atrophy, visual disturbances, fatigue, vertigo, and convulsions. Death is sometimes caused by cerebral hemorrhage which in turn may be due to infarction or rupture of a vessel or even to hypertension.

Articular symptoms and skin involvement are frequent. Articular symptoms include both transitory arthralgia and migratory arthritis, as well as myalgia resembling the muscle pain and soreness of trichinosis. Many types of skin lesions have been observed, some of the most common of which are erythema, petechiae, purpura, and especially subcutaneous nodules. The skin overlying the subcutaneous nodules often displays red or violet discolorations, and ulceration, hemorrhage and gangrene are sometimes noted. A special cutaneous form of the disease characterized by cutaneous infarc-

tions with necrosis has been observed. It is usually unaccompanied by systemic manifestations, and appears to run a more benign course.

The mortality rate is high but the disease is not invariably fatal. It may run from acute to chronic stages and range anywhere from a few days to as long as several years in length. Periods of remission may occur, especially when the patient is being treated, but even in remission, although there is an absence of symptoms, no histologic healing of the lesions already present takes place. A few cases of apparent recovery have been reported, probably in cases in which there was a localization of the disease or a minimal of organs involved. However, a long term follow-up is necessary to prove permanent recovery.

This inflammatory disease of the arterial system involves all layers of the small and medium sized arteries and arterioles. There is a primary injury to the vessel wall, accompanied by swelling and fibrinoid degeneration of the media with edema and a thready fibrinous exudate, resulting in a slight reduction in the lumen of the vessel. This is followed by an inflammatory stage in which there is an infiltration of the media and adventitia with polymorphonuclear neutrophils, eosinophils, lymphocytes, and plasma cells. This results in destruction of the internal elastic membrane as well as necrosis of the media which may give rise to thrombosis or aneurysm formation. If these aneurysms rupture, hemorrhage will occur. The further marked proliferation of fibroblasts from the adventitia results in a partial or complete closure of the lumen. This occlusion of the vessel interferes with the blood supply to the various organs and thus produces the observed degeneration and infarction in the tissues.

The laboratory findings vary from person to person depending on the organs involved and the degree of involvement. There is usually a moderate to marked leukocytosis with an occasional increase in polymorphonuclear neutrophils. Eosinophilia is sometimes marked, mostly when there is an associated allergic manifestation such as asthma. According to Allen, Barker, and Hines, eosinophilia occurs in less than 20% of cases, but when present may be very pronounced. A mild to severe normocytic anemia and rapid erythrocyte sedimentation rate occur, especially during the active phase of the disease. Demonstration of a lupus erythematosus cell is sometimes possible.

Bloody stools may result when the gastrointestinal tract is involved, and urinary findings such as hematuria, albuminuria, red and white blood cells and casts indicate renal involvement. There is also an increased plasma globulin and hypoproteinemia, with x-ray and electrocardiograph changes corresponding to the degree of pulmonary and heart involvement.

Diagnosis, until recently, was seldom made until necropsy. Now a clearer understanding of the clinical and pathologic features of the disease makes diagnosis ante mortem more likely. Often diagnosis is not diffi-



cult on clinical grounds when the symptoms are typical. However, when cases simulate other connective tissue disorders, remembering that in periarteritis nodosa, renal, cardiovascular, neurological, and gastrointestinal manifestations predominate is helpful.

It is generally thought that periarteritis should be suspected in a patient who has an obscure type of illness characterized by fever, progressive decline of general health, and symptoms and signs suggesting the presence of a diffuse systemic process. The presence of the following would aid in diagnosis although many of these are not specific for this particular disease: leucocytosis, rapid erythrocyte sedimentation rate, hypertension, and especially failure of the condition to respond to the conventional treatment. An absolute and final diagnosis can be made only by histologic examination of a subcutaneous nodule or muscle tissue. Even then, although a positive result shows the presence of the disease, one negative result does not exclude the diagnosis. More than one area may have to be subjected to biopsy before the diagnosis is definitely established.

Until the advent of steroids, there was no definite treatment which either prevented or appreciably affected the course of periarteritis nodosa. In the past, various drugs such as antihistamines, antisyphilitic drugs, sulfonamides, penicillin, and para-amino-salicylic acid were used but to little avail. Symptomatic therapy directed toward the specific tissues or organs involved was and still is used but offers no cure. For example, Demerol® and codeine may be used to relieve severe pain, aspirin to relieve myalgia, and blood transfusions to compensate for the blood loss or anemia.

Recently cortisone, corticotrophin (ACTH) and various other steroids have been used to bring symptomatic relief and sometimes a temporary remission of the disease. The suppression of symptoms by steroids is largely due to the way they affect the body. They act as anti-inflammatory agents and suppressants of antibody production. Furthermore, they suppress tissue damage from antigen-antibody interaction. Doses of the hormones are usually quite large at first, and are then regulated according to the patients' responses. After administration of steroids, the fever often subsides within 24 to 72 hours, the symptoms appear somewhat less severe, and the erythrocyte sedimentation rate in some patients gradually decreases. Partial relapse occurs in most cases after withdrawal of the hormone; when the treatment is resumed these cases improve again. Despite this apparent relief, biopsy taken after treatment will still show the presence of lesions. In fact, healing of lesions does not usually occur, but if it does, occlusion of the vessel results and may produce widespread visceral infarction. So, although steroids may provide symptomatic relief and prolong life, they offer no cure.

#### ILLUSTRATIVE CASE

A 20-year-old white female was admitted, complaining of abdominal pain and nausea of four to five days duration. Eight months previous to admission and again two weeks prior to

admission she was given antibiotics for a sore throat. Since the first time she had felt slowed down and tired.

When she was admitted she was found to have a soft apical systolic murmur and numerous petechiae on the skin and mucous membranes. Her symptoms by this time included a steady backache, abdominal pain, bloody diarrhea, joint pains, tachycardia, fever, and hypertension.

The laboratory work revealed a marked normocytic anemia, albuminuria and gross hematuria, bloody stools, moderate leucocytosis as well as hypoproteinemia with an increase in plasma globulin. The blood urea nitrogen was also elevated. A bone marrow smear revealed excessive granulopoiesis consistent with infection and a skin and muscle biopsy revealed focal vasculitis of the dermis.

#### TREATMENT

Treatment consisted of multiple transfusions to control the marked anemia, codeine to relieve pain, and several antibiotics. When the patients condition steadily declined, the steroids — prednisone and hydrocortisone — were employed. The hematuria and bloody diarrhea subsided on steroids, and her temperature returned to normal. Her condition continued to improve until the abrupt onset of generalized convulsions, which were accompanied by dyspnea, pulmonary edema, and heart failure. On autopsy, lesions and areas of hemorrhage were found in the heart, lungs, gastrointestinal tract, and kidneys and were diagnosed as periarteritis nodosa.

The above case illustrates the general symptoms and widespread involvements of the organs of the body in periarteritis nodosa. This disease of unknown etiology may be treated by massive doses of steroids, which may induce clinical remissions but rarely alter the fatal course.

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# Folic Acid Deficiency In Pregnancy: A Sorely Neglected Clinical Entity

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Anemia of pregnancy due to folic acid deficiency is common, often fulminating and severe, potentially fatal, and infrequently diagnosed and treated. Its occurrence in our affluent society is just as shocking as would be a case of florid rickets or scurvy. The hemoglobin may drop abruptly to 2-3 grams /100 ml. because of hemolysis, with a concomitant leucopenia and thrombocytopenia. Delivery is therefore hazardous. Folic acid deficiency is a major cause of abruptio placentae.<sup>1,2</sup>

There has not been general awareness of this entity because folic acid assays have not been widely used and bone marrow aspirations are done infrequently in pregnancy. The peripheral blood is usually not macrocytic, although often becomes so after a concomitant iron deficiency is corrected. The marrow may not be megaloblastic, and the diagnosis is often missed even when an intensive study of this anemia is being carried out.<sup>3</sup> A megaloblastic marrow may return to normal within hours after therapy, including a good hospital diet, or after delivery alone. The typical megaloblastic marrow may appear only some time after delivery<sup>4</sup> as in case No. 1. Folic acid deficiency is the most likely cause of third trimester anemia, particularly if iron therapy has been used and the anemia develops rapidly.

Giles and Shuttleworth<sup>5</sup> report folic acid deficiency in one of thirty-nine pregnancies in an average British population. They express surprise at this high incidence, but other authors believe the incidence may be higher.<sup>3</sup> The British diet seems to be low in folic acid-containing foods, although, as discussed below, derangements of folic acid metabolism occur in spite of a seemingly adequate diet.

Six cases of anemia of late pregnancy and the early puerperium were observed at the Eastern Maine General Hospital within a six-month period. Three were definitely megaloblastic anemia, two were probable folic acid deficiency, and a sixth is reported as possible folic acid deficiency. A seventh definite case was recently observed. Four of the patients were private patients of one obstetrician, confirming the fact that the incidence of this anemia is a function of the index of suspicion of the clinician.<sup>5</sup> None of the seven patients received folic acid prenatally. Clinics elsewhere have observed an

increased incidence because of the apparently ill-advised removal of folic acid from prenatal capsules.

Case No. 1: This 20-year-old white, para 3, gravida 3 housewife has led a nomadic existence in humble circumstances for several years with a grossly inadequate diet. She had only the sketchiest prenatal care. The first delivery in 1958 was uneventful. She had abscesses of the back and groin, and the discharge hematocrit of 31% was not explained.

In May, 1962, she was admitted at term with her second pregnancy which had been complicated by heartburn and edema. The hematocrit was 8%, R.B.C. 800,000/mm<sup>3</sup>, W.B.C. 4,200/mm<sup>3</sup> with many large multilobed polymorphonuclear leukocytes in the 71% segmented population. Platelets 23,000/mm<sup>3</sup>. Reticulocytes 0.3%. The bone marrow showed a marked depression of the erythroid series, only 2.5% of the total nucleated count. A few could be recognized as possibly megaloblastic. The metamyelocyte and stab forms were unusually large, and the marrow neutrophils were hypersegmented as in the peripheral smear.

It is of interest that one pathologist reviewed the marrow and suggested the possibility of myelogenous leukemia. (See discussion, case No. 6)

Rapid clinical improvement and elevation of the reticulocyte count occurred with oral folic acid therapy.

The third admission for a full term delivery occurred August 1963. The hematocrit was 23%; she was transfused at time of delivery, and discharged without study. She had no prenatal care, but during pregnancy she suspected she was again anemic and sent her husband to a practitioner for some iron-containing tablets. Two months postpartum she was again admitted to the hospital with severe weakness and pallor. The hematocrit was 7%, hemoglobin 2.2 grams/100ml., R.B.C. 625,000/mm<sup>3</sup>, W.B.C. 3,100/mm<sup>3</sup>. The platelets appeared increased. The bone marrow was markedly megaloblastic with half the nucleated cells in the red cell series showing abnormalities consistent with megaloblastic arrest. Again a shift to the left in the granulocyte series and giant stab forms were noted.

She was transfused three times; there was a clinical and reticulocyte response to folic acid orally, and when last seen in the O.P.D. eight months postpartum, the hematocrit was 39%.

DISCUSSION: This is a most disturbing case, for this completely preventable serious anemia occurred twice and possibly three times. One cannot be critical of the physicians involved, for it is only recently that there has been a profusion of articles on folic acid deficiency in pregnancy. There was no general realization that once a megaloblastic anemia of pregnancy occurs, the chances are better than 50% of its recurring without folic acid prophylaxis.<sup>6</sup> Such a diagnosis should be indicated in big red letters on the chart as is done with patients sensitive to penicillin.

A unit record system (combined hospital-clinic charts) would probably not have prevented this occurrence with this unintelligent nomadic patient. But any patient can be taught to repeat parrot-fashion "severe folic acid anemia" and perhaps alert the next obstetrician.

Case No. 2: A 20-year old white primipara with an uneventful full term delivery. There had been moderate nausea and anorexia the first trimester of pregnancy. Diet appeared adequate,

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the history being obtained from her husband, a candidate for a doctorate in nutrition. Her solicitous father-in-law, a drug-gist, loaded her up with hundreds of prenatal capsules, none containing folic acid. The hemoglobin was 10.0 grams/100 ml. Two months antepartum and at delivery the hematocrit was 30% and the hemoglobin 10.0 grams/100 ml. These values rose to 35% and 11.9 gms/100 ml. following transfusion, but two days later the hematocrit dropped to 25% with no significant blood loss. W.B.C. 8,500/mm.<sup>3</sup> Reticulocytes 5.2%. Platelets 281,000/mm.<sup>3</sup> The marrow two days postpartum showed a megaloblastic arrest in the red cell series with 29% of the total nucleated count represented by these abnormal forms. There was no shift to the left in the granulocyte series. The response to folic acid was satisfactory.

DISCUSSION: Folic acid deficiency must be considered in well-to-do private patients and not just the poor. Our cases do not bear out the statement that anorexia, gastrointestinal disturbances, and failure to maintain weight are essential features.

Case No. 3: A 19-year-old multipara who had been delivered elsewhere six days p.t.a. Two previous pregnancies and prenatal periods had been normal, as was the dietary history. There was moderate blood loss at delivery but none since. The hematocrit three days postpartum was 17%, and on admission was 7% with a W.B.C. of 10,300/mm.<sup>3</sup> reticulocytes 0.5%, and platelets with 171,000/mm.<sup>3</sup> In spite of her alarming blood picture she was alert and conscious and not in shock. She was transfused six times with a resulting hematocrit of 34%. A marrow obtained after transfusion showed a megaloblastic maturation abnormality of the red cell series, these constituting 42% of the total nucleated count. A shift to the left of the granulocytes was noted as was a paucity of stainable iron in the hyperplastic marrow fragments. She was discharged in apparent good health, and a follow-up was not obtained.

DISCUSSION: Folic acid deficiency is the commonest cause of a fulminating anemia late in pregnancy or puerperium.

Case No. 4: A 15-year-old unmarried white primipara was admitted in active labor. There was no prenatal care. Her father was a woodsman with 17 children. Several individuals elicited a dietary history of almost exclusively carbohydrates. (This was probably correct, since on the hospital diet sheets she routinely checked only mashed potatoes.) The admission hematocrit was 28%, and two days later was 24% with a hemoglobin of 7.9 grams/100 ml. W.B.C. 12,200/mm.<sup>3</sup> Platelets appeared adequate on smear. The marrow was obtained 5 days postpartum, during which time she had been persuaded to eat a well rounded hospital diet. The marrow was hyperactive but with no evidence of megaloblastic arrest. There was in fact an erythroid depression with red cells accounting for only 12% of the total nucleated count. Metamyelocytes (and less mature cells) accounted for 30% of the granulocytes. There was no stained iron in the hyperplastic marrow fragments.

A reticulocyte rise occurred four days after folic acid administration, and she was discharged with a hematocrit of 30% on the 12th hospital day. She did not return for follow-up.

DISCUSSION: There is no proof that this patient suffered from folic acid deficiency, but she was "guilty until proven innocent." The complex series of events that resulted in this patient having no definitive therapy for five days will not be recounted. This was a serious error, since cases No. 1 and No. 3 show the manner in which the hemoglobin can drop suddenly to 2-3 grams/100ml. One problem is that the physician orders a routine blood examination and in the normal course of events may not see the report for 48 hours. Obstetrical nurses and laboratory technicians should be impressed with the gravity of the appearance of an anemia on the obstetrical wards or clinic. The appropriate clinician or the pathologist should be notified promptly.

Case No. 5: A 20-year-old white primipara with a normal

prenatal course except for a hemoglobin of 10.0 grams/100 ml. during the seventh month of pregnancy. There was no significant loss of blood at delivery, but two days postpartum the hematocrit was 25%. Reticulocytes 3.8%. The marrow was mixed with peripheral blood but of average cellularity with no iron stainable in the fragments.

DISCUSSION: Again there is no proof of folic acid deficiency, but it would have been reprehensible not to treat her with folic acid. The diagnosis can often not be made with a single marrow.<sup>3</sup>

Case No. 6: A 31-year-old white multipara whose hemoglobin at time of delivery was 13.2 grams/100 ml. Her prenatal course and delivery were uneventful. There was no history of any type of blood loss, but eight days postpartum she complained of dizziness and numbness of the extremities. On readmission the hematocrit was 10% and the hemoglobin 3.5 grams/100 ml. W.B.C. 32,400/mm.<sup>3</sup> with marked shift to the left. The drop in hemoglobin had apparently occurred in about three days, since a postpartum hemoglobin was 10.0 grams/100 ml. The serum bilirubin was 2.9 mg/100 ml with a partition of 1.0/1.9. The stool guaiac was 4 plus. (This case is not being reported in detail.) The marrow prior to transfusion was markedly hyperplastic with no evidence of maturation derangement. The RBC's accounted for 34% of the total nucleated count. Granulocytes younger than band forms accounted for 29% of the total nucleated cell count. Again there was no stainable iron. Reticulocytes 14%.

DISCUSSION: Obviously this entire picture could be accounted for by gastrointestinal bleeding, although x-ray study of the G.I. tract was negative. Again it would have been reprehensible to withhold folic acid. The marrow of case No. 1 at one time also suggested leukemia. We have observed bizarre blood pictures in other patients with folic acid deficiency, and the leukemoid blood picture has been reported in folic acid deficiency of pregnancy. Three cases of leukemoid reaction were seen in three months in a small British Hospital.<sup>6</sup> This case was discussed with Dr. Victor Herbert who felt that it could have been either iron deficiency or folic acid deficiency. The fulminating character of the anemia suggests the latter, and folic acid deficiency can cause bleeding as in abruptio placentae.

## DISCUSSION OF PROBLEM

*Folic acid metabolism:* This complex subject, including formation and destruction of folic acid in the gut, will not be discussed. But the fact that folic acid deficiency of pregnancy may develop with a seemingly adequate diet demands some explanation. Folic acid metabolism is deranged by a variety of factors. The best known, but probably not the most common, is deficiency of vitamin B12. Hence the identical blood picture of folic acid deficiency and pernicious anemia. Drugs such as Dilantin,<sup>®</sup> Butazolidin,<sup>®</sup> and alcohol may produce a megaloblastic blood picture. There is speculation that toxins from urinary infections of pregnancy or even some as yet undiscovered humoral factor in pregnancy may derange folic acid metabolism.

The most important factor is that the body stores of folic acid are limited, in contrast to iron and vitamin B12. Total deprivation of the latter two in the diet would not produce signs of deficiency for several years. On a folic acid free diet, clinical and laboratory evidence of deficiency may occur within a month. Dr. Victor Herbert maintained himself on such a diet, but objective evidence of deficiency took somewhat longer to appear. It is of interest that he noted mental dullness



at the end of the trial period, and Pritchard<sup>4</sup> noted similar findings in his pregnant women. As with iron, folic acid is parasitized by the fetus; hence the deficiency in the mother. Folic acid deficiency occurs in infants but not apparently as a result of maternal deficiency.

Folic acid deficiency has a hemolytic component, a fact of importance since other hemolytic anemias occur in pregnancy. Megaloblastic marrows occur frequently with hemolytic anemias<sup>7</sup> because overactive erythropoiesis quickly depletes the stores of folic acid. Thus, a vicious cycle is established. Many chronic hemolytic anemias respond well to folic acid and are in difficulty less frequently on folic acid maintenance.

There is recent strong evidence that spontaneous abortions, early in pregnancy, are often associated with folic acid deficiency.

*Diagnosis of folic acid deficiency:* A megaloblastic marrow in a pregnant woman in an age group where pernicious anemia is rare is probably but not necessarily due to folic acid deficiency. The shortcomings of a single postpartum marrow and the peripheral blood smear have been noted. The reticulocyte response to folic acid therapy in medical patients is difficult to evaluate unless the patient has been kept on a folic acid free diet, a procedure that is hard on both the patient and the dietitian. It would be most inappropriate to do this except on an experimental basis in postpartum patients. The value of the FIGLU test in pregnancy is controversial, chiefly because folic acid is metabolized by the fetus and formiminoglutamic acid does not accumulate. Serum vitamin B12 levels, if normal, exclude pernicious anemia but may be low in folic acid deficiency. Folic acid assays of the serum are rarely available, and even if they are, there is real urgency in starting therapy with folic acid.

*Prophylaxis and therapy of folic acid deficiency of pregnancy:* Because of the difficulty of establishing the diagnosis in many cases and the gravity of the condition, the clinician must treat on suspicion. The average daily requirements of folic acid are probably 25mcg. but are higher in pregnancy. Prenatal capsules containing 1 mg. of folic acid are adequate prophylaxis. The usual therapeutic dose of 15 to 20 mg daily would be far in excess of requirements except for the fact that folic acid deficiency causes alterations in the small intestine that make for malabsorption of the vitamin.

The hazard of treating pernicious anemia with folic acid is minimal in the childbearing age group. "Pernicious anemia of pregnancy" has been considered identical to true pernicious anemia because it was not realized that folic acid deficiency responds briefly and with a reticulocyte response to moderate doses of vitamin B12.

Our cases bring out the frequent occurrence of concomitant iron deficiency, a factor in the prevention of the typical macrocytic peripheral blood picture.

#### SUMMARY

The authors would not ordinarily rush into print

with a series of cases in half of which the diagnosis has not definitely been established. The necessity of sounding a warning on this severe and potentially fatal anemia prompted us to do so. Private and clinic patients should have prophylactic folic acid administration. Many prenatal capsules now contain no folic acid.

At the Eastern Maine General Hospital, all pregnant clinic patients with a hemoglobin of 10.0 grams/100 ml. or less have a medical consultation; there is evidence that the figure perhaps ought to be 11.0 grams/100 ml. Significant anemias of the third trimester or puerperium are most likely due to folic acid deficiency, particularly if it develops rapidly for no obvious cause. The blood picture may be bizarre, possibly suggesting leukemia.

Clinicians are often loath to administer folic acid unless the blood picture is classical. For about fifteen years the hazard of administering folic acid to pernicious anemia patients has been dinned into us so that the use of folic acid seems almost malpractice.

The problem of an anemia is often similar to that of acute sepsis. In the latter situation vigorous efforts are made to isolate an organism. But if this cannot be done and the patient's condition continues grave, an educated guess as to the best antibiotic therapy is made. If the anemia is suspected of being megaloblastic a medical emergency exists. A marrow and such appropriate baseline studies as can be done at that particular hospital should be carried out quickly and vigorous therapy, possible of both folic acid and vitamin B12, should be started. At a later date pernicious anemia can be ruled out.

Since there is certain to be inertia in reestablishing routine prenatal folic acid administration and since many women will continue to receive no prenatal care, the physician's index of suspicion for folic acid deficiency in pregnancy should remain high. Internists should be aware of derangements of folic acid metabolism since they explain several other baffling anemias.

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# Use of Polystyrene Particles in Serodiagnosis†

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Polystyrene particles have currently a wide application in the fields of clinical and veterinary medicine. Their use is an application of immunological principles designed to lead to the visual detection of antigen and antibody reactions. Polystyrene particles are recent in a series of particles used for this purpose. Some of their antecedents have included red cells, and particles of siliceous earth with fairly uniform size, for example, Wyoming bentonite.<sup>11</sup> The technique of preparing the latex particles so that they will function in a serological test for the purpose of specific antigen or antibody are covered in various textbooks. Oreskes et al<sup>10</sup> discuss in some detail the mechanism of particulate carrier techniques. It may suffice to consider the following simple processes. A homogenous protein is selected and injected into an animal of a foreign species, as for example, crystalline egg albumin injected into a rabbit. After multiple injections the serum can be harvested and contains a high concentration of antibodies to the injected protein. Latex particles of uniform size then may be exposed to the antibody containing serum and the antibody protein absorbed onto the surface of the particles. These so called sensitized particles can be properly diluted until they maintain a homogenous dispersion. Subsequently, in the presence of specific antigen, an antigen-antibody reaction occurs and the result is to agglutinate the particles, disturbing the stability of the dispersion, with the development of aggregates of particles which are then visible to the naked eye.

**RHEUMATOID ARTHRITIS:** A widespread use of sensitized polystyrene particles has been the serological diagnosis of rheumatoid arthritis.<sup>1,2,3,4</sup> The rheumatoid factor is a protein, a gamma globulin with a molecular weight of 900 000. Whether the rheumatoid factor consists of only one specific protein or an aggregate of several is not clear. The rheumatoid factor is usually present in the serum of patients with rheumatoid arthritis. It also occurs in a wide variety of other illnesses but with less frequency.

Several applications utilizing polystyrene particles have been employed. In one situation polystyrene particles coated with gamma globulin are caused to agglutinate when serum containing the rheumatoid factor is added.<sup>1,2</sup> In another, uncoated particles are also presumably actually agglutinated when the rheumatoid

factor is present. The fine agglutination can be appreciated if eosin is added to the mixture.<sup>3</sup>

Various reports indicate that from 70 to 90% of sera from patients with rheumatoid arthritis react positively, and the incidence of false positive reactions in non-arthritic persons is said to be about 2%.

**PREGNANCY TESTS:** A prototype of the test used to determine pregnancy<sup>16,20,21</sup> utilizes two reagents. The first is a serum containing a quantity of antihuman chorionic gonadotrophin antibodies obtained by the injection of purified human chorionic gonadotrophin into rabbits. The second reagent is a suspension of latex particles upon which have been absorbed human chorionic gonadotrophin molecules. An appropriately concentrated sample of urine is mixed with the antibody reagent. The second reagent is subsequently added to the mixture. If agglutination of the sensitized particles occurs, it is presumed that the urine contained an insufficient amount of chorionic gonadotrophin to react with and bind, in antigen-antibody complexes, the antihuman chorionic gonadotrophin in the first reagent; and hence a negative test. If, however, agglutination does not occur; it is presumed that the antihuman chorionic gonadotrophin was bound by a quantity of gonadotrophin in the urine; hence a positive test.

This test has been shown to be reliable, superior to biological assay using toads and frogs, and equal in reliability to biological assays utilizing hyperemia of the rat ovary as an end point. Convenience and rapidity of the test performance, coupled with the freedom from biological factors which complicate the responses in test animals in the biological assays would appear to make these tests preferable. They are, of course, no more useful than biological assays in early diagnosis, or in differentiating tubal pregnancies from other pelvic disorders. Their reactivity depends upon achieving the same minimal urinary concentration of gonadotrophin.

**C-REACTIVE PROTEIN:** The C-reactive protein is an abnormal protein moiety associated with the lipids of human serum and found in persons with a great variety of inflammatory diseases, both infectious and noninfectious.<sup>12,13</sup> It has the capacity to precipitate specifically a polysaccharide extracted from diplococcus pneumoniae.<sup>19</sup> The substance is referred to as the C, for carbohydrate, reactive protein. The polystyrene reagent in this instance is prepared by absorbing serum which is hyperimmune to the C-reactive protein onto the surface of polystyrene particles. Serum containing C-reactive protein in which the complement has been deactivated by heating and which has been properly diluted, causes macroscopic clumping when mixed with these particles.

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**THYROID DISEASE:** For some time it has been known that circulating antibodies to thyroglobulin exist in many patients.<sup>15</sup> It is presumed that when thyroglobulin is released in the tissue as a result of a number of possible biological incidents, including surgical injury, infection, damage due to the change associated with hyperplasia and involution, that in those persons prone to develop auto immune disease, antibodies to the thyroid globulin will be formed. There is relatively poor correlation between the type of disease and the presence of the auto immune substance.<sup>17</sup> Briefly, approximately 10% of people without demonstrable thyroid disease show circulating antithyroglobulin, a slightly larger number with miscellaneous thyroid disorders, and a peak incidence in people with chronic thyroiditis.

The test material is prepared by exposing latex particles to concentrations of purified human thyroglobulin. Antithyroglobulin antibodies present in the serum of suspect patients are then detected by macroscopic agglutination.

**LUPUS ERYTHEMATOSUS:** In evaluating test procedures for lupus erythematosus it should be born in mind that in the hands of different investigators LE cell preparations are positive in well documented disease in varying incidences. This ranges from 60% to nearly all and may bear some relation to the vigor with which efforts to find the LE cells phenomena are prosecuted. In any event one could devoutly wish that a more simplified procedure could be devised to detect the presence of antinuclear antibody. However, it appears that the sensitized polystyrene particle does not fill the bill.<sup>22</sup> Only about a third of the people with clinically distinct lupus erythematosus who also have a positive LE cell preparation will also have polystyrene particle agglutination.

The preparation of sensitized particles is done by exposing the polystyrene units to a substance derived from calf thymus which is rich in highly polymerized desoxy-ribose nucleic acid. The antigen antibody reaction and subsequent agglutination of the sensitized particles, is an indication that antinuclear antibody, postulated in people with lupus erythematosus, is present.

**FIBRINOGEN:** Particles sensitized by fibrinogen are reacted with human plasma and macroscopic agglutination occurs when fibrinogen is present. Tests can be roughly quantitated by observing the intensity of the agglutination, and the test has also the advantage of speed of performance and the exclusion of the technique for determining clotting time.

**GAMMA GLOBULIN:** A screening test for the presence of gamma globulin has been devised. In this instance the polystyrene particles have been sensitized by exposure to a dispersion of antibodies to human gamma globulin which have been produced in rabbits by the injection of electrophoretically pure gamma globulin. In the presence of gamma globulin the sensitized particles are agglutinated. The test can be roughly quantitated by dilutions of the serum being tested. We have

not felt that the screening test was useful because it tended to produce estimates of gamma globulin consistently greater than the electrophoretically determined fraction. This has suggested that there might be a cross reaction among the other globulins of the serum. Since the detection of lower levels of gamma globulin will be missed because of this, an electrophoretic separation should probably be utilized in gamma globulin deficiency suspects. In interpreting the test in infants it should be recalled that infants characteristically pass through a physiological state of hypogammaglobulinemia during their first year of life.<sup>23</sup>

**VETERINARY APPLICATIONS:** Polystyrene particles have an ongoing importance in the field of veterinary medicine, both from a practical and from an investigational point of view.<sup>7</sup> Experimentally, workers in the veterinary field are applying polystyrene particles tests to serological diagnosis of numerous bacterial, fungal and viral diseases, including specifically such disorders as leptospirosis, tularemia, mycobacterium infections and salmonellosis.

Several disorders have implications in both veterinary and clinical medicine. One of these is histoplasmosis, a fungus infection, for which a useful serological test has been devised.<sup>5</sup> Some of the diagnostic difficulties incurred in the use of skin testing, that is terminal anergy, or the widespread incidence of positive skin tests in persons not apparently affected, may be circumvented by this test which is easier to perform, in this instance, as in all of the occasions under discussion, than the traditional serological procedures.

**TRICHINOSIS** is another disorder of concern to veterinarians and physicians. The polystyrene test for this disease utilizes an antigen which is a boiled alkaline saline extract of acetone dried trichinella larvae. It is the impression of workers in the field that this slide test is useful in detecting specific antibodies in nearly every instance in which the complement fixation test has detected antibodies, and at times, sooner than the complement fixation test.<sup>8</sup>

Hydatid disease<sup>9</sup> and brucellosis<sup>6</sup> are diseases in which agglutination of sensitized polystyrene particles has been useful in serodiagnosis.

#### SUMMARY

Without attempting to review exhaustively all of the recent and current applications of this new technique, we have presented, together with critical observations when they appeared indicated, some situations in which it has appeared to be useful. It is a new and reliable tool in the armamentarium of the serologist which should prove increasingly helpful because of its ease of performance and interpretation.

#### REFERENCES

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*Continued on Page 234*

# Endometrial Carcinoma

ALICE J. SHUBERT, M.D.

During the years 1956 through 1963, a seven year period, there were 79 cases of endometrial carcinoma reported at the Eastern Maine General Hospital. Of this total 64 were living, according to the 1963 check-up survey. Among the 17 who died, seven expired because of metastatic disease, all of which were diagnosed in advanced stages. One patient died of carcinoma of the breast, discovered in the physical examination, at time of treatment for endometrial carcinoma. The remainder of the deaths were due to cardiovascular difficulties per se or cardiovascular complicated by diabetes.

The following chart shows the survival rate by years.

Year	Total No. Treated	No. Living	No. Dead
1956	7	1	6
1957	9	6	3
1958	10	8	2
1959	14	12	2
1960	8	7	1
1961	11	11	0
1962	13	10	3
1963	7	7	0

In reviewing the charts, three of the 79, were not available, so the following figures are based on a total of 76 patients.

Age groups:

Years	—	35-39	40-44	45-49	50-54	55-59
		1	1	7	12	13
Years	—	60-64	65-69	70-74	75-79	80-84
		15	16	8	2	1

The above figures show the peak incidence ages to be in the 50-70 year age groups. The average age of the patients diagnosed was 60.5. Only one of the 76 women had a history of previous radiation treatment for benign disease.

Vaginal bleeding from spotting to profuse flow, was the chief complaint of most patients. The duration varied from three days to as long as ten years in one patient who at the time of diagnosis and treatment had advanced carcinomatosis.

Twelve patients had associated fibroids. In two cases the hospitalization was for hysterectomy because of fibroids. The diagnosis of adenocarcinoma of the endometrium was made postoperatively.

Two patients were hospitalized for D & C's because of bleeding cervical polyps. Laboratory diagnosis of adenocarcinoma was obtained and hysterectomy followed in each case.

According to the pathologic staging by Javert and Hafamann Stage O, cancer limited to the endomet-

rium, Stage I, myometrial invasion only, Stage II, pelvic spread to adnexa and upper vagina, Stage III, regional lymphatic metastasis and Stage IV, hematogenous metastasis, carcinomatosis, these 76 patients could be grouped as follows:

Stage	Number of Patients
O	10
I	53
II	7
III	1
IV	5

This staging was determined from pathological findings and may not be entirely accurate.

Type of treatment varied depending upon the surgeon's choice and the extent of the disease. Many authors state that preoperative insertion of radium followed by a hysterectomy is the treatment of choice. Javert and Renning-Cancer, 16:1057 (August) 1963 state that the trend is away from the procedure of choice. These authors write that in the past ten years the method of choice used with increasing frequency is primary hysterectomy, followed by X-Ray therapy if there has been deep penetration of the disease into the muscular layer and/or to the serosa. Primary hysterectomy alone is done if only superficial invasion has occurred.

In this hospital the procedure of choice has been primary hysterectomy as shown by the following figures.

- Hysterectomy — 49
- Hysterectomy plus X-Ray — 10
- Radium Insertion plus Hysterectomy — 11
- X-Ray plus Hysterectomy — 1
- X-Ray — 1
- Radium plus X-Ray — 1
- Radical Hysterectomy — 1

No Treatment — 2 — These patients expired from cardiovascular complications before treatment could be started. Each had a diagnostic D & C to establish the diagnosis of endometrial carcinoma.

In reference to hysterectomy as treatment — a pan-hysterectomy as well as bilateral salpingo-oophorectomy was always performed.

The above findings bear out the obvious conclusions of Javert that when the cancer is confined to the uterus, radiation alone, surgery alone, or combinations of the two, will frequently cure the patient, but when the cancer has gone beyond the uterus, these modalities no longer produce satisfactory results.



## Clinico-Pathological Conference

A 61-year-old farmer was admitted to the Eastern Maine General Hospital at 7:25 p.m. on January 23, 1964 because of shortness of breath and fever of approximately 12 hours' duration.

His first admission to this hospital was on May 11, 1962 for a tracheotomy because of progressive obstruction of his airway. While attending the funeral of his brother, who died in 1959 of coronary occlusion, the patient is said to have "passed out," fallen to the floor and to have remained unconscious for several minutes. The exact etiology of this episode was not determined. He is said to have had shortness of breath since this episode.

In 1960 the patient is said to have had an attack of "viral pneumonia" associated with which there was an acceleration of his dyspnea accompanied by cough and fever. The acute illness lasted several days and then subsided, but he remained more dyspneic than he had been previously. From this time on he had gradually increasing shortness of breath on exertion, and he had frequent choking spells. His wife described severe respiratory noises when he was asleep.

Physical examination (5-11-62) showed a pleasant 59-year-old man with marked stridor and exertional dyspnea. His ears, nose, and pharynx appeared normal. There was a bilateral paralysis of the vocal cords with both cords in the adductal position. The heart was not enlarged. There were no murmurs. The chest was clear to auscultation and percussion. His blood pressure was 162/114 mm.Hg. His temperature was 98.6° F.; pulse, 80/min.; and respirations, 24/min.

Under local anesthesia a routine tracheotomy was done using a vertical incision; a No. 5 tracheotomy tube was inserted. His postoperative course was uneventful; his symptoms were relieved and he was discharged improved on May 14, 1962 with a diagnosis of stenosis of the larynx due to abductor paralysis (probably central).

About two months after his tracheotomy, after spreading a large amount of fertilizer, he became extremely dyspneic at 3:00 a.m. and was admitted to another hos-

pital where he was treated with oxygen, suction, and sedation for what was assumed to be an irritative bronchitis. He has not really felt well since that time.

His second admission to the Eastern Maine General Hospital was on September 12, 1962 because of increasing shortness of breath especially on exertion, frequently accompanied by choking spells. His wife has described severe respiratory noises when he is asleep. The patient has no known allergies. Despite his severe dyspnea he has missed very little time at work and is now caring for more livestock than ever. He feels that hay is especially apt to produce dyspnea, but he has noted no particular relation of dyspnea to entering the barn or to exposure to silage. He had pneumonia as a child during the influenza epidemic of 1918. There is no history of rheumatic fever. He has had no edema. He sleeps well; he does not use alcohol; he stopped smoking 10 years ago. About two weeks ago he took digitalis for four days. It was stopped because of the development of a fluttering sensation in his chest. He recently lost 8 or 9 pounds following diuretic therapy. He is taking no medication at present.

Physical examination (9-12-62) shows a well-developed, well-nourished male in no distress. Temperature, 98.6°F; pulse, 78; respirations, 20; blood pressure 150/90 mm.Hg. (both arms, sitting). Head, negative. Extra ocular movements are normal. Pupils and conjunctivae are normal. The optic discs are clear. There is moderate arteriolar narrowing and variation in the light reflex. The ears and nose and throat are negative. The thyroid is not palpable. The tracheotomy tube is in place with minimal reaction around it. There is no general glandular enlargement. The chest expands well. There is some limitation of motion in the diaphragms but they are not immobile. There are occasional rhonchi which clear on coughing. The lungs are otherwise clear. There is no prolongation of expiration nor is there use of the accessory muscles of respiration. The heart is not enlarged. The sounds are of good quality. A<sub>2</sub> is louder than P<sub>2</sub>. There are no murmurs. The liver is palpable 2cm below the right costal margin. The genitalia are

LABORATORY STUDIES: Kahl, Hinton, and V.D.R.L. are negative.

[illegible]

	<i>Transaminase</i>	<i>LDH</i>
1-23-64	32	590
1-24-64	16	540

not remarkable. Rectal examination is negative except for a 2 plus normal range of generalized prostatic enlargement. There are no nodules. The extremities show a normal range of motion. There is no clubbing, cyanosis, or edema. The peripheral pulses are palpable and equal. The deep tendon reflexes are active and equal. Sensory examination is normal.

URINES:

<i>Date</i>	<i>pH</i>	<i>Sp. Grav.</i>	<i>Alb.</i>	<i>Sugar</i>	<i>Acetone</i>	<i>WBC</i>	<i>RBC</i>	<i>Casts</i>
5-12-62	7.5	1.019	0	0	0	rare	0	0
9-12-64	7.5	1.010	0	0	0	1-3	0	0
1-22-63	5.5	1.012	0	0	0	rare	0	0
11-11-63	7.5	1.019	0.01	0	0	3-6	4-8	0
1-23-64	7.5	1.005		0	0			
1-24-64	6.0	1.005	0.01	0	0	0-4	4-8	0

ECG: (9-12-62) Conduction disturbance; otherwise within normal limits — (1-22-63) Axis normal, but has shifted approximately 30° to the right from tracing of 9-12-62. (from plus 30° to plus 60°). Probably within normal limits. Slight axis shift of questionable significance — (10-31-63) Sinus tachycardia, minimal ST segment changes since the previous tracing, probably not of diagnostic significance. Axis is similar to that of 9-12-62 — (11-11-63) Slowing of the heart rate since the previous tracing, slight interventricular conduction disturbance. No significant change from the previous ECG.

2-21-63 — PULMONARY VENTILATION STUDIES  
(New England Deaconess Hospital)  
AGE: 60 years HEIGHT: 64 inches WEIGHT: 179 pounds  
BODY SURFACE AREA: 1.86  
PREOPERATIVE DIAGNOSIS: Pulmonary emphysema  
POSTOPERATIVE DIAGNOSIS: Pulmonary emphysema and Fibrosis  
VITAL CAPACITY: Measured: 3000 ml  
Predicted: 3430 ml  
% of Predicted: 91%  
TIMED VITAL CAPACITY: 1 second 2000 (66%); 2 second 2500 (83%); 3 second 2800 (93%)  
MAXIMUM BREATHING CAPACITY: Measured 21.6 L/min.; Predicted 23 L/min.  
WALKING VENTILATION: 14.3 L/min.

DISCUSSION: The maximum breathing capacity was unsatisfactory because tracheotomy valve could not follow the fast excursions. Timed vital capacity shows only minimal obstruction.

VENTILATING INSUFFICIENCY: Arterial blood gas analysis at rest, exercise, and breathing 100% oxygen were as follows:

	<i>O<sub>2</sub> tension</i>	<i>pH</i>	<i>CO<sub>2</sub></i>	<i>pCO<sub>2</sub></i>
Rest	100	7.48	24	31
Exercise	99	7.42	24	37
Oxygen	101.6	7.47	23	31

IMPRESSION: Hyperventilation. Mild obstructive respiratory insufficiency. X-rays: (B-4929) *Chest* (9-12-62). The heart and great vessels are within normal limits of size and shape. The lung fields are clear without change since the previous examination of 9-10-62. The tracheotomy tube remains in good position.

(9-13-62) Fluoroscopy of the chest reveals normal action of both leaves of the diaphragm with excursion of both leaves well within the normal limits of variation. The heart is not remarkable for the age; nor is the aorta. No masses can be seen in the mediastinum, films of the chest taken in the PA view in deep inspiration and deep expiration disclose at least

a travel of 1½ ribs of the right leaf and a little more on the left. There is no paradoxical motion which would indicate paralysis of either leaf of the diaphragm.

(1-19-63) Negative roentgen examination of the chest without change since the previous examination. (with tracheotomy tube still in position).

(1-20-63) Negative roentgen examination of the chest, tube still in position.

(1-21-63) There is a suggestion of a small density in the left upper lung field at the level of the 2nd anterior interspace which was not apparent on films of 1-7-63 and might represent a new process of uncertain etiology. The area is

small but certainly this could represent a small area of pneumonia or even a small infarct as clinically suggested.

(1-24-63) There seems to have been a resolution of the process previously described in the left upper mid-lung field. Certainly the density which was visible on those films is no longer definitely demonstrated. There are a few more linear type densities in this area, which could represent the dissolving process in this same general area. The type of resolution would suggest more the possibility of a resolving pneumonia than an infarct.

(1-28-63) Since the previous examination there has been complete resolution of the small density in the left upper mid-lung field. The chest now appears normal except for the presence of the tracheotomy tube.

(10-31-63) Since the previous films the patient has developed some density in the right lower lung field in the PA views and has the appearance of some pneumonia and in part possibly embolus as clinically suggested. This area was completely clear on the previous films of 1-28-63 and now, of course, is unclear. The left lung looks generally the same as here. The metallic tracheotomy tube is in place. The heart remains unenlarged.

(11-6-63) Since the previous examination there has been complete clearing of the previously described pulmonary infarctions. The left lung is clear. The metal tracheotomy tube remains in position in the trachea.

The patient did quite well from the time of his admission 9-12-62, and had no dyspnea during this hospitalization. Because of the questionable nature of his dyspnea he was taken on a walk of three flights of stairs accompanied by his physician and on this exertion there was minimal dyspnea. It was not considered to be marked. The presence of a low CO<sub>2</sub> combining power suggested the possibility of some hyperventilation. Arrangements were made to do oxygen saturations and tensions as well as CO<sub>2</sub> tension. Phenobarbitol was prescribed, and the patient was discharged on 9-18-62.

After discharge from the hospital, pulmonary function studies showed pH, 7.48; a pO<sub>2</sub>, of 68 mm.Hg., and a pCO<sub>2</sub> of 37.5 mm.Hg. The question of diffusion block or ventilation perfusion imbalance was raised. As a result of the pulmonary function studies this diagnosis could be neither substantiated nor denied. It was felt



that many of his symptoms were indeed related to anxiety. He did quite well from September through December, 1962.

His *third admission* was on 1-19-63 because of dyspnea of 24 hours' duration. For approximately two weeks prior to admission he had noted gradually increasing shortness of breath on exertion. He had had no difficulty at night, but on walking to the barn or walking out of doors at all or doing any sort of moderate exercises such as cleaning the milking machine his dyspnea becomes sufficiently marked to make him stop carrying out such chores. During the 24 hours prior to admission his dyspnea greatly increased and appears most marked after walking or working in the barn. There has been no chest pain, orthopnea, paroxysmal nocturnal dyspnea or ankle edema. Skin tests by an allergist showed the patient to be allergic to a number of substances. Desensitization shots were in progress at the time of his present admission. Although much of the patient's dyspnea occurs while he is in the barn, it is felt that the dyspnea is related specifically to exertion rather than to exposure to irritating substances in the barn. The patient has had no chills, fever, cough or sputum since his episode of "viral pneumonia" in 1960. There is no history of edema, leg pain, or phlebitis.

Except for his dyspnea the patient has felt well. His appetite has been good. He has been extremely apprehensive and anxious about his physical condition and also about his farm which, although well maintained during his illness has needed more attention and time than he has been able to give it.

Physical examination (1-19-63) revealed a slightly obese, dyspneic male with cyanosis of the lips and questionable cyanosis of the nail beds. His respirations are rapid (26) and quite deep. Temperature 98.6°F; pulse 90; and regular; blood pressure 116/80 mm.Hg. The chest expands well. The diaphragms descend normally. The breath sounds are minimally increased throughout and there are no rales or rhonchi. There is no prolongation of the expiratory phase of respirations nor is there use of accessory muscles of respiration. The heart is not enlarged to percussion. The sounds are of good quality, A<sub>2</sub> equals P<sub>2</sub>. No murmurs are heard. The liver is palpable 2cm below the right costal margin and is non-tender. No spleen or kidneys are palpable. A hard movable nodule is palpable in the subcutaneous tissue of the abdominal right upper quadrant. There is no superficial engorgement or distention of the veins of the extremities. There is no calf tenderness. Homan's sign is negative. Peripheral pulses are palpable and equal in the lower extremities. The deep tendon reflexes are active and equal.

The patient was given oxygen by catheter through his tracheotomy tube at seven liters per minute, (bubbled through water). He was put on a 500mg sodium diet. A progress note in the chart suggests that the chest films of 1-19-63 show poor aeration compared with some of his earlier films. The patient responded

well to conservative treatment. His temperature which rose to 100.8°F. on 1-20-63 dropped to normal the next day and stayed within normal range for the rest of his hospitalization. A medical consultant (1-26-63) reviewed the history and physical findings on this patient suggesting that whatever organic disease might be present the symptoms of the patient were greatly exaggerated by psychiatric components. He suggested studies to rule out the possibility of methemoglobinemia being a factor in the production of this clinical syndrome. The patient was discharged on 1-19-63.

In February, 1963, the patient was sent to the Lahey Clinic for pulmonary function studies because of apparent progression of asthma or his pulmonary symptoms. There the diagnosis of emphysema and asthma were made. No other cause for the dyspnea was found. Multiple allergies were found and desensitization therapy was instituted. During the summer of 1963 he did fairly well but in October, 1963, he noted increasing tiredness, weakness, and shortness of breath.

His *fourth* Eastern Maine General Hospital admission was on 10-31-63 because of chills and cough. That morning he awoke early with a shaking chill followed by an episode of coughing which produced a small amount of blood-streaked sputum. There was associated nausea and he vomited his breakfast. He was seen in the Accident Room by his physician shortly after this episode. At that time there were audible rales at the right base and the patient was admitted to the hospital for further evaluation. During recent weeks the patient has experienced a sensation of obstruction in the low substernal region when he swallowed. There is no history of blood, melena, black or tarry stools.

Physical examination (10-31-63) reveals a dyspneic but not cyanotic male in no acute distress, lying quietly in bed and with a temperature of 101°F.; pulse, 110; respirations, 28; and blood pressure of 110/70 mm.Hg. His tracheotomy is functioning well. His chest expands well. There are fine inspiratory rales at the right base. There is no dullness. The left lung reveals no abnormalities. The superficial leg veins are not distended. There is no calf tenderness, or fullness. Homan's sign is negative. There is no popliteal or adductor canal tenderness. The deep tendon reflexes are active and equal. Weight is 183 pounds.

He was started on penicillin, 500,000 units every three hours and heparin 100mg, i.m. every eight hours. At the end of 48 hours his temperature had dropped to 98.6°F. and it remained within normal range for the rest of his hospitalization, his pulse fluctuated between 118 and 80 for the first four days and his respirations varied from 32 to 18 and his blood pressure showed moderate fluctuation between 122/90 to 156/100 mm.Hg. A penicillin resistant staphylococcus was isolated from his sputum on 11-2-63. Chloromycetin,<sup>®</sup> 250mg, four times a day was added to his therapy. The patient responded to treatment. The anticoagulant therapy was changed from heparin to Coumadin.<sup>®</sup> On 11-13-63 he was discharged



improved to continue on long-term Coumadin therapy on an out-patient basis.

His *fifth admission* was on 12-22-63 because of diarrhea. During the previous two or three weeks he had had intermittent episodes of diarrhea characterized by 10 to 15 loose watery stools per day without bleeding. He was unable to cope with a continued seepage from the rectum. A fecal impaction was removed in the Accident Room. He had also noticed a decrease in the size of his urinary stream. A retrograde pyelogram was negative and a barium enema was negative except for possible polyps in the large bowel. A suprapubic prostatectomy was done on 1-2-64 on which day he received one unit of whole blood. His post-operative course was uneventful, and he was discharged improved on 1-18-64.

His *sixth admission* was on 1-23-64 because of shortness of breath. His long-term anticoagulant therapy had not yet been reestablished. He had been quite well until the morning of 1-23-64 when he noted shortness of breath which increased throughout the day and during the afternoon associated with a fever of 103°F. There was no associated chills and no associated chest pain. Physical examination (1-23-64) reveals a somewhat cyanotic, dyspneic, slightly obese male with a tracheotomy tube in place and functioning well. His temperature was 102.2°F; pulse, 120; respirations, 16; blood pressure, 130/90 mm.Hg. The chest expands well. There is no prolongation of expiration but the respirations are deep and somewhat labored. No rales, rhonchi, or dullness are noted. The heart is not enlarged. The sounds are of good quality. No murmurs are heard. There is a sinus tachycardia. The extremities show a normal range of motion. There is no clubbing, cyanosis, or edema.

He was given heparin, 100mg. subcutaneously (deep) every 8 hours. Oxygen was administered by catheter through the tracheotomy tube at six liters per minute. He was also given phenobarbitol. On 1-24-64, the patient appeared definitely improved. His temperature dropped to 99°F; his pulse to 98 and his blood pressure to 110/70 mm.Hg. His respirations rose to 24/min, and he raised a moderate amount of sputum which was occasionally blood-streaked. During the early hours (1:00-3:00 a.m.) on 1-25-64, his respirations were quite noisy but later he appeared to be sleeping quietly. He expired at 8:16 a.m. on 1-25-64.

#### DIFFERENTIAL DIAGNOSIS: Harold D. Cross, M.D.

The main symptoms this patient experienced recurrently were dyspnea and anxiety. The dyspnea apparently dated back to about 1959 or 1960 and was associated with minimal exertion, even with eating. It was exacerbated by a "viral pneumonia" in 1960. No cause for the dyspnea was found until he was seen by an ENT Specialist in May, 1962 at which time the vocal cords were found fixed in the midline and the patient had marked stridor. This was a bilateral abductor paralysis. Following tracheotomy the patient was considerably improved, and gained an estimated 70 pounds over the next year. At

the time of laryngoscopy, no other motor, sensory, or mechanical abnormality such as fixation of the arytenoids was found. The tracheotomy tube employed was a flutter-valve type such that he exhaled through the larynx and thus could speak.

In spite of the above symptomatic and objective improvement, the patient continued to be dyspneic, with periodic exacerbations. In January, 1963, he was first described as cyanotic and for the first time an abnormal patchy density was seen on chest X-ray. This was thought to be pneumonic or embolic. Similar episodes occurred in October, 1963 and January, 1964; the latter terminating with his death. There was associated cough and hemoptysis on at least two occasions. At no time, however, were any signs of phlebitis detected. He was anticoagulated for about one month in November, 1963, with apparent improvement. This was discontinued because of impending prostatic surgery for an obstructive prostate. He had been off anticoagulants about one month when the terminal hospitalization occurred.

In order to account for such a discrete motor paralysis, with no abnormality or surgery of the neck or mediastinal structures, the lesion would have to be in the medulla, in the lower portion of the nucleus ambiguus. This motor tract is located fairly laterally and inferiorly in the medulla, the vagal fibers leave this area by traversing dorsally then laterally and inferiorly. There is no decussation. It is immediately apparent, knowing these anatomical locations and courses, that any single lesion causing bilateral involvement would have to be quite large — at least occupying more than half the cross-diameter of the medulla, to affect both nuclei. It is inconceivable that this would occur without producing other symptoms. Therefore I believe that neoplastic disease is ruled out. Also hemorrhagic or thrombotic disease is similarly excluded in that more than one vessel would have to be affected to produce such a bilaterally symmetrical paralysis. The only other type of vascular lesion is that of vasculitis. This seems unlikely, in the sense of picking these structures only, but is a possibility. The diagnosis of lead poisoning, various plant or vegetable poisons is beyond proof or definition so I will leave that. Infectious processes — especially syphilis is ruled out by the serology. The static nature of the process is puzzling, that is, how such a clear-cut isolated paralysis could remain without further evidence of disease, leaves me with more unanswered questions.

As to the nature of the pulmonary problem, I am not able to make any definite diagnosis from the data presented. Pulmonary function tests in October, 1962 showed on oxygen tension of 68mm.Hg. which corresponds to a saturation of approximately 94%. Those done at Lahey Clinic in February, 1963 showed saturations somewhat higher than is physically possible, which illustrates a problem with regard to the technique of determining blood oxygen concentration in this portion of the oxy-hemoglobin dissociation curve. With the low pCO<sub>2</sub>, slightly high pH and ventilatory studies very close



to normal, all one can say is that he was hyperventilating, apparently chronically. It would have been interesting, and possibly of some help clinically, to have repeated these studies when he had an exacerbation of his illness.  $p\text{CO}_2$  alveolar-arteriolar gradients of more than the usual 1-2 mm.Hg. are reported in pulmonary emboli. Also exaggeration of the hypoxia would be present (cyanosis was described).

In summary, I believe the patient had some type of medullary disease affecting both nuclei ambigu, possibly a vasculitis, and that he had multiple pulmonary emboli with or without infarctions and infection.

#### **PATHOLOGIC DISCUSSION: Richard C. Wadsworth, M.D.**

First, I should like to congratulate Doctor Cross for his discerning discussion of an extremely complicated and clinically baffling case. His conclusion regarding the anatomic location of the pathologic lesion is substantiated by the histologic findings.

Necropsy revealed a moderately obese adult male measuring 65½ inches in length. An open remote tracheotomy wound was observed in the anterior neck. A recent healed suprapubic surgical scar showed no demonstrable drainage. As the necropsy incision was carried down through the suprapubic region there was noted a cyst-like cavity filled with oily, reddish-brown, slightly turbid fluid lying between the subcutaneous fat and the left rectus muscle. A culture of this fluid revealed *Aerobacter aerogenes*, non-hemolytic streptococci and a coagulase-positive, penicillin-sensitive staphylococcus aureus. Incidentally, a post-mortem blood culture was sterile.

The heart weighed 330gms. The pericardial surfaces, valves, and chambers were not remarkable. The coronary arteries and the first portion of the aorta showed moderate arteriosclerotic changes. The coronary arteries were narrowed with lipid deposition, calcium deposition, and hemorrhage in the thickened intima, but no demonstrable occlusion. The myocardium showed only occasional small irregular areas of fibrosis. No sharply demarcated areas of infarction were demonstrated.

The left and right lungs weighed 246 and 313gms. respectively. Both lungs revealed small patchy areas of atelectasis and moderate congestion. No emboli could be demonstrated either grossly or microscopically. There was no demonstrable pneumonic process. A small focal area of pulmonary adenomatosis was demonstrated microscopically in a section from the right lower lobe.

The spleen, weighing 353gms, showed moderate congestion.

The pancreas was not remarkable.

The esophageal mucosa showed scattered plaque-like thickenings in the distal third. Microscopically there were focal areas of hyperplasia of the stratified squamous, epithelium and small areas of epithelial erosion accompanied by an infiltration of lymphocytes and plasma cells in the underlying stroma. The rest of the gastrointestinal tract was not remarkable.

The gall bladder contained approximately fifty small black stones but showed no evidence of inflammation or neoplasm.

The liver weighed 1,845gms and appeared large without grossly demonstrable abnormalities. Occasional vacuoles could be demonstrated in the liver cells. There was a slight periportal lymphocytic infiltration.

Both adrenals showed slight nodular, cortical hyperplasia.

The combined weight of the kidneys was 326gms. There were two cortical cysts in the right kidney. Both kidneys were moderately congested. The capsular spaces and convoluted tubules contained red cells and eosinophilic coagulated protein. Well formed hyaline casts were demonstrable in the lumina of the loops of Henle and the collecting tubules. The cortical cysts contained eosinophilic coagulated protein. Some nephrosclerosis was demonstrable in the medium-sized arteries. The bladder showed focal areas of congestion and lymphocytic infiltration. A spermatocele of the left epididymis measured 2.2cms in diameter. The testes were not remarkable.

The thyroid gland weighed 24gms. Microscopic sections were not remarkable.

The vertebral bone marrow was slightly hyperplastic.

The larynx contained a moderate amount of brown, somewhat tenacious mucoid material. The laryngeal mucosa was covered by well-differentiated stratified squamous epithelium. There were scattered focal accumulations of lymphocytes in the underlying loose connective tissue stroma. A few scattered lymphocytes were demonstrable between the adjacent striated muscle fibers.

The brain weighed 1,355gms. There was no evidence of extradural, subdural, or subarachnoid hemorrhage. The cerebral hemispheres appeared symmetrical without flattening of the convolutions. Multiple horizontal sections through the brain and cervical spinal cord showed no gross abnormalities. The microscopic examination of the central nervous system revealed lesions in the medulla oblongata and in the upper cervical cord. At the level of the exit of the glossopharyngeal nerve were found small bilaterally symmetrical lesions in the region of the nucleus ambiguus. These lesions had a granulomatous appearance and consisted of necrotic foci filled with epithelioid cells and lymphocytes. Other sections of the medulla and cervical spinal cord revealed perivascular lymphocytic cuffing and an occasional area of recent perivascular hemorrhage. Sections from the motor cortex, basal ganglia, and temporal lobes were not remarkable.

Spontaneous bilateral abductor paralysis of the vocal cords is not a common occurrence. When it does occur, it is quite apt to be fatal. A tracheotomy can be life-saving in these people. Although anatomic lesions symmetrically located in the upper medulla were demonstrable in this patient, the pathogenesis of the lesions is not readily explained. There appears to be a sequence of events which may be helpful in reconstructing the

pathogenesis of this lesion. His dyspnea appears to have started in 1959, slightly more than three years before his death, and there appeared to be a temporal relationship with the funeral of his brother who is said to have died of coronary artery disease. An attack of "viral pneumonia" in 1960 was accompanied by marked acceleration of his dyspnea and frequent choking spells. The bilateral abductor paralysis of the vocal cords was first verified in May of 1962.

The central nervous system pathology observed in the post-mortem examination appears to be limited in distribution to the medulla and upper cervical spinal cord. The predominant abnormality found was a perivascular lymphocytic cuffing in these areas. The demonstrable tissue destruction was in the medulla with nearly symmetrical involvement of the nuclei ambiguus. The type and distribution of the lesions suggests a regional encephalitis of probable virus etiology, and bears some resemblance

to the idiopathic focal encephalitis of the brain stem described by Kavanaugh and Goldstein<sup>1</sup> and called by them acute idiopathic metencephalitis. They point out that this disease is frequently associated with mild infection of the upper part of the respiratory system or "grippe-like" symptoms. Two similar cases were reported by Walker<sup>2</sup> in 1953.

#### ANATOMICAL DIAGNOSIS

- Idiopathic focal encephalitis of brain stem
- Abscess of anterior abdominal wall (post-prostatectomy)
- Moderate coronary atherosclerosis

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DEAN H. FISHER, M.D.  
COMMISSIONER

State Of Maine

## Department of Health and Welfare

### Vitamins – Use and Misuse

ELLA LANGER, M.D.\* and DOROTHY WOODCOCK\*\*

Vitamins<sup>1</sup> have long been recognized as organic substances which the body needs for metabolism, but does not synthesize in sufficient quantities. As a result, such substances must be supplied by food source or by artificial means to meet the needs of the individual. In the past, the normal food supply was not adequate to meet the needs of vitamin D for children and adolescents.

Over forty years ago Dr. Edward Mellanby<sup>2</sup> demonstrated that cod liver oil would prevent rickets. Since then rickets, caused by vitamin D deficiency, formerly a serious problem in this area, has become a rare clinical entity in temperate climates but does still occur in this country, Great Britain, Canada, and elsewhere.

The pendulum has now made a full swing from the period prior to Dr. Mellanby's discovery. We are now fearful that people of all ages will consume too many vitamins from food supplemented with vitamins and from commercial vitamins. This is disturbing in view of our knowledge that a well-balanced diet prepared by approved methods from foods found in our markets supplies all of the vitamins needed by the average child or adult, and at much less cost than do commercial vitamins. Families or individuals who purchase expensive multi-vitamins, frequently take from their food money to do so.

As more is learned about the inter-relation of vitamins and other nutrients, the possibility of developing imbalance of body chemistry becomes more apparent and the recognition that there is much that is not known about the inter-relation of nutrients in the human body becomes clear. Further, there is a very real danger of people depending on commercial sources of vitamins to solve their health problems instead of the family doctor. Our special concern is directed to children and their consumption of fat soluble vitamins. It is now evident that even the small infant who consumes milk, margarine and cereal with vitamin D added, may receive the total recommended intake of vitamin D, thus making it unnecessary, and in fact undesirable, for him to take an additional source of vitamin D in view of the

undesirable sequelae caused by an excess of vitamins, especially fat soluble vitamins.

Conversely, as Dr. Arthur Lesser reminds us,<sup>3</sup> it must also be kept in mind that many children do not have milk with vitamin D added, including those who consume plain pasteurized milk, raw milk, and reconstituted dry skim milk. To make certain that each and every child is receiving enough and not too much vitamin D it is necessary to ascertain his total intake of vitamin D in food, including milk and in any commercial vitamins.

Further, the child who appears hungry and malnourished is not necessarily going to benefit from vitamin D supplement of the diet.

A few leading questions, (or at times a complete diet history), will often help to determine just what nutrients may be deficient in a particular child's diet; calories, protein, calcium, iron or what vitamins other than vitamin D may be needed. Additional food as can be provided through the Special Milk and School Lunch Programs and the Donated Food Program for Needy Families may provide the required nutrients. Guidance regarding good food and nutrition practices is being provided by public health nurses, extension workers and others. Nutrition guidance for a particular patient or patient's family could be arranged for on referral to the Nutrition Services, Division of Maternal and Child Health.

Children for whom vitamin supplements have been ordered should be checked periodically to determine that such vitamins are still needed. In the event that the family who have been giving their children reconstituted dry skim milk have an increase in income and start using homogenized milk, their children will probably no longer need vitamin D supplementation, nor will they need other vitamins if they are consuming a well-balanced diet and if extra vitamins are not indicated for a medical reason.

Further, it is evident that vitamin D is needed in certain amounts by children of all ages, and adolescents to help to maintain a normal calcium-phosphorus metabolism.

The addition of vitamin D (the only known vitamin not present in the adequate diet in sufficient amounts

\*Director, Division of Maternal and Child Health

\*\*Nutrition Consultant

for the normal person at any age) to the commercial milk supply provides a logical method for Americans, especially children, to receive this nutrient. The addition of vitamin D to other foods seems unnecessary, and because of variable food habits, a relatively unsure way of good distribution of this needed nutrient.

The National Research Council recommends 400 International Units of vitamin D for pregnant women and children through the age of 18; 1500 International Units of vitamin A are recommended for infants, with a gradual increase in amount to 5000 International Units for children in the 12 to 18 age group and adults; 1000 additional International Units are advised for pregnant women in the second and third trimesters, and 3000 additional for lactating mothers.

The following up-to-date resource of nutrition materials, prepared for Maine people having a normal diet, may be obtained from this office on request:

#### MAINE DIET MANUAL — Revised 1963

(a) Normal diets — pages 3 through 14

Children's leaflets — ABC'S OF GOOD EATING, HELPING YOUR CHILD TO EAT WELL, A GUIDE TO FOOD PLANNING FOR OUR CHILDREN, FOOD FOR TEEN-AGE GIRLS AND BOYS, THE GOOD BOX LUNCH, LIVER FOR YOUR HEALTH.

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## Dr. Langer Retires

After twenty years of yeoman service within the Department, Dr. Ella Langer, Director of the Division of Maternal and Child Health and Crippled Children's Services, has retired. She came to Maine in 1944 to serve as assistant to the director of Crippled Children's Services and the next year became director of the two programs comprising the Division of Maternal and Child Health and Crippled Children's Services which post she has filled with distinction over the period of her service with the Department.

Dr. Langer graduated from the University of Vienna Medical School following which she was the resident physician at the University's Children's Hospital for 3 years. During that period, she also served as director of Public Health Services in a rural district of lower Austria. During the years in which she was school physician for the City of Vienna, she pursued the practice of private pediatrics. Awarded three scholarships by the Commonwealth Fund in New York City for the study of public health, she followed this course in England, Scotland, France, Holland and Switzerland — also, at the Academy of Social Hygiene in Dusseldorf, Germany.

With the annexation of Austria, Dr. Langer emigrated to New York and took the New York Medical Examiners Board examination for accreditation in this country. Shortly, she was offered a position as research fellow at Johns Hopkins Medical School where she took part in a group study on the prevention of deafness in

school children, for a period of 3 years. The following year she spent at the Fels Research Institute for Child Growth and Development at Antioch College, Yellow Springs, Ohio, as research pediatrician. Returning again to Baltimore, she led a busy life as part-time health officer with the City Health Department, part-time school physician, working with Dr. Helen Taussig on rheumatic fever and congenital heart conditions and conducting her private practice in a housing project for war workers.

Among many other extra-curricula activities, Dr. Langer served as visiting lecturer on maternal and child health at the Harvard School of Public Health from 1955-1959 and as pediatrician on the Honorary and Consulting staff of the Central Maine General Hospital in Lewiston. She is a diplomate of the American Board of Pediatrics as well as the American Board of Preventive Medicine. She has received many honors and accolades during her years of service in Maine, among the latest, a plaque recently presented to her by the Pine Tree Society for Crippled Children and Adults and a citation for service from the Maine Rehabilitation Association. Perhaps her highest award, however, is the affection held for her by her many colleagues in and outside of her chosen profession.

Dr. Langer states that although her future plans are somewhat indefinite, she does expect to continue her Maine residence and to serve, on a part-time basis at least, as a Pediatric Consultant.





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## *Maine Heart Association Notes*



### **The Effect Of Digitalis Upon The Exercise Electrocardiogram**

"The electrocardiographic exercise test is being used with increasing frequency as a means of detecting coronary artery disease and in evaluating work capacity in patients with known cardiac lesions. Occasionally, the test is employed in patients who are receiving digitalis preparations. . . . The following study was undertaken to evaluate the effect of digitalis upon the ECG exercise test in normal subjects and in patients with heart disease. . . .

"Electrocardiographic exercise tests were performed in 15 patients and 16 normal subjects before and after full digitalizing doses of digoxin. While receiving digoxin, 14 of 15 patients and 8 of 16 normal subjects developed "positive" tests. Five of the 15 patients and all of the normal subjects had "negative" tests when not receiving digoxin.

"Although the mechanism of the effect of digoxin upon the ECG exercise test remains unknown, no evidence was found to suggest that myocardial ischemia was produced by the drug. An effect upon intramyocardial potassium seems to be most likely. ECG exercise tests should not be carried out in patients who are receiving digitalis preparations. Digitalis should be discontinued for at least three weeks before an exercise test is performed."

Submitted by Jacob B. Dana, M.D.

Reference: Kawai, C. and Hultgren, H. American Heart Journal, Volume 68, pages 409-419, 1964.

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### **"After A Coronary"**

The above is the title of a new publication by the American Heart Association to help dispel many of the fears and worries of a patient recovering from a heart attack. In the leaflet some of the matters that puzzle and worry patients are discussed in layman's language. The discussion is intended to supplement the physicians counseling and put the patients mind at rest on some of the intangibles of his condition. The leaflet has been published for distribution by physicians and copies can be obtained, free of charge, by writing the Maine Heart Association, 116 State Street, Augusta, Maine.



# County Society Notes

## PENOBSCOT

A meeting of the Penobscot County Medical Society was held at The Oronoka Restaurant in Orono, Maine on October 20, 1964. The meeting was conducted by the President, William A. Purinton, M.D.

Dean Fisher, M.D., Commissioner of the Department of Health and Welfare, State of Maine, was the guest speaker. He discussed the Kerr-Mills Bill and other methods of handling the indigent patient's medical expenses. A lively discussion period was held following his lecture.

HADLEY PARROT, M.D.  
*Secretary*

## SOMERSET

A meeting of the Somerset County Medical Society was held on October 20, 1964 at the Oak Pond Restaurant in Canaan, Maine. The meeting was called to order by H. Carl Amrein, M.D., Vice-President.

Following several minor items of business, the matter of appointment of a representative from the society to contact members who have been or are delinquent in paying the \$25.00 assessment for the Maine Medical Education Foundation was brought to the attention of the society. After some discussion, the motion was made and seconded that the secretary write to the Maine Medical Association to the effect that the society would not appoint any member to "police" other members. It was felt that the matter of paying the assessment was a personal one.

Harland G. Turner, M.D. was appointed chairman of the County Diabetes Committee by the President, W. Edward Jordan, Jr., M.D.

Thomas A. Martin, M.D., President of the Maine Medical Association, was present as guest and spoke on political issues, both within and outside of the state, with reference to the medical profession.

The meeting was adjourned and a buffet luncheon was enjoyed by the members.

MARIAN L. STRICKLAND, M.D.  
*Secretary*

## HANCOCK

A meeting of the Hancock County Medical Society was held on November 11, 1964 at the Hancock House in Ellsworth, Maine.

Rudolf E. Eyerer, M.D., Pathologist at the Eastern Maine General Hospital, was the guest speaker. He spoke on the problems and techniques of interpretation of Papanicolaou's smears.

RUSSELL G. WILLIAMSON, M.D.  
*Secretary*

## New Member

### PENOBSCOT

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USE  
CHRISTMAS  
SEALS

1964 Christmas

1964 Greetings

Fight Tuberculosis  
and other  
Respiratory Diseases









APR 1 1964

# MAINE MEDICAL ASSOCIATION

## *OFFICIAL ROSTER*

### Association Members

#### County and Alphabetical Listing

Supplement to

The Journal of the Maine Medical Association

Volume 55, Number 3

March, 1964

# Past Presidents

## Maine Medical Association

*Isaac Lincoln, M.D., Brunswick	April-June, 1853	*Stanley P. Warren, M.D., Portland	1911-1912
*James McKeen, M.D., Topsham	1853-1854	*Ralph H. Marsh, M.D., Guilford	1912-1913
*Charles Millett, M.D., Lewiston	1854-1855	*W. C. Peters, M.D., Bangor	1913-1914
*Joseph H. Estabrook, M.D., Camden	1855-1856	*H. L. Bartlett, M.D., Norway	1914-1915
*Hosea Rich, M.D., Bangor	1856-1857	*Erastus E. Holt, M.D., Portland	1915-1916
*Gilman Daveis, M.D., Portland	1857-1858	*W. F. Hart, M.D., Camden	1916-1917
*J. C. Bradbury, M.D., Old Town	1858-1859	*James A. Spalding, M.D., Portland	1917-1918
*H. H. Hill, M.D., Augusta	1859-1860	*George H. Coombs, M.D., Waldoboro	1918-1919
*T. G. Stockbridge, M.D., Bath	1860-1861	*H. B. Mason, M.D., Calais	1919-1920
*H. M. Harlow, M.D., Augusta	1861-1862	*Theodore E. Hardy, M.D., Waterville	1920-1921
*Alonzo Garcelon, M.D., Lewiston	1862-1863	*Addison S. Thayer, M.D., Portland	1921-1922
*J. T. Gilman, M.D., Portland	1863-1864	*L. T. Snipe, M.D., Bath	1922-1923
*N. P. Monroe, M.D., Belfast	1864-1865	*C. A. Moulton, M.D., Harland	1923-1924
*Amos Nourse, M.D., Bath	1865-1866	*F. W. Mann, M.D., Houlton	1924-1925
*S. H. Tewksbury, M.D., Portland	1866-1867	*J. D. Phillips, M.D., Southwest Harbor	1925-1926
*Cyrus Briggs, M.D., Augusta	1867-1868	*L. P. Gerrish, M.D., Lisbon Falls	1926-1927
*I. T. Dana, M.D., Portland	1868-1869	*Herbert F. Twitchell, M.D., Portland	1927-1928
*D. McRuer, M.D., Bangor	1869-1870	*Frank Y. Gilbert, M.D., Portland	1928-1929
*B. F. Buxton, M.D., Warren	1870-1871	*Delbert M. Stewart, M.D., South Paris	1929-1930
*A. J. Fuller, M.D., Bath	1871-1872	*Charles B. Sylvester, M.D., Portland	1930-1931
*A. P. Snow, M.D., Winthrop	1872-1873	*Ernest V. Call, M.D., Lewiston	1931-1932
*A. F. Page, M.D., Bucksport	1873-1874	*E. Delmont Merrill, M.D., Dover-Foxcroft	1932-1933
*Thomas H. Brown, M.D., Paris	1874-1875	Warren E. Kershner, M.D., Bath	1933-1934
*J. H. Bates, M.D., Yarmouth	1875-1876	*Edwin W. Gehring, M.D., Portland	1934-1935
*E. F. Sanger, M.D., Bangor	1876-1877	*John L. Johnson, M.D., Bangor	1935-1936
*T. H. Jewett, M.D., South Berwick	1877-1878	Frederick T. Hill, M.D., Waterville	1936-1937
*M. C. Wedgwood, M.D., Lewiston	1878-1879	*Ralph W. Wakefield, M.D., Bar Harbor	1937-1938
*S. C. Gordon, M.D., Portland	1879-1880	Willard H. Bunker, M.D., York Harbor	1938-1939
*William Warren Greene, M.D., Portland	1880-1881	George L. Pratt, M.D., Farmington	1939-1940
*A. K. P. Meserve, M.D., Buxton	1881-1882	Thomas A. Foster, M.D., Portland	1940-1941
*George E. Brickett, M.D., Augusta	1882-1883	*P. L. B. Ebbett, M.D., Houlton	1941-1942
*Oren A. Horr, M.D., Lewiston	1883-1884	Carl H. Stevens, M.D., Belfast	1942-1943
*Thomas A. Foster, M.D., Portland	1884-1885	*Oscar F. Larson, M.D., Machias	1943-1944
*Sumner Laughton, M.D., Bangor	1885-1886	*R. V. N. Bliss, M.D., Blue Hill	1944-1945
*J. B. Walker, M.D., Thomaston	1886-1887	*Adam P. Leighton, M.D., Portland	1945-1946
*Frederick C. Thayer, M.D., Waterville	1887-1888	*John O. Piper, M.D., Waterville	1946-1947
*Stephen H. Weeks, M.D., Portland	1888-1889	Stephen A. Cobb, M.D., Sanford	1947-1948
*Benjamin F. Sturgis, M.D., Auburn	1889-1890	Forrest B. Ames, M.D., Bangor	1948-1949
*Samuel B. Hunter, M.D., Machias	1890-1891	Ralph A. Goodwin, M.D., Auburn	1949-1950
*Edwin M. Fuller, M.D., Bath	1891-1892	Foster C. Small, M.D., Belfast	1950-1951
*Alfred Mitchell, M.D., Brunswick	1892-1893	C. Harold Jameson, M.D., Rockland	1951-1952
*John A. Donovan, M.D., Lewiston	1893-1894	*Eugene H. Drake, M.D., Portland	1952-1953
*W. P. Giddings, M.D., Gardiner	1894-1895	Norman H. Nickerson, M.D., Greenville	1953-1954
*Lewis W. Pendleton, M.D., Portland	1895-1896	*Robert W. Belknap, M.D., Damariscotta	
*D. A. Robinson, M.D., Bangor	1896-1897	June-August 1954 (Died in Office)	
*Wallace K. Oakes, M.D., Auburn	1897-1898	William F. Mahaney, M.D., Saco	1954-1955
*Charles O. Hunt, M.D., Portland	1898-1899	Martyn A. Vickers, M.D., Bangor	1955-1956
*Bigelow T. Sanborn, M.D., Augusta	1899-1900	Armand Albert, M.D., Van Buren	1956-1957
*Edward H. Hill, M.D., Lewiston	1900-1901	Francis A. Winchenbach, M.D., Bath	1957-1958
*Frederic H. Gerrish, M.D., Portland	1901-1902	Eugene E. O'Donnell, M.D., Portland	1958-1959
*Hiram Hunt, M.D., Greenville	1902-1903	Allan Woodcock, M.D., Bangor	1959-1960
*Augustus S. Thayer, M.D., Portland	1903-1904	*Wilson H. McWethy, M.D., Augusta	
*F. L. Dixon, M.D., Lewiston	1904-1905	June 1960-February 1961 (Died in Office)	
*Randall D. Bibber, M.D., Bath	1905-1906	Carl E. Richards, M.D., Sanford	February 1961-June 1961
*C. E. Williams, M.D., Auburn	1906-1907	James A. MacDougall, M.D., Rumford	1961-1962
*B. B. Foster, M.D., Portland	1907-1908	Ralph C. Stuart, M.D., Guilford	1963-1964
*Alfred D. Sawyer, M.D., Fort Fairfield	1908-1909		
*Galen M. Woodcock, M.D., Bangor	1909-1910		
*E. H. Bennett, M.D., Lubec	1910-1911		

\* Deceased



# Members

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(Corrected to February 1, 1964)

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*President* — Robert D. Wakefield, M.D.

*Secretary-Treasurer* — Donald L. Anderson, M.D.

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Amfilo, Basil	626 Main St., Lewiston
Anderson, Donald L.	369 Main St., Lewiston
Anderson, Dorothy	369 Main St., Lewiston
Archambault, Philip L.	346 Main St., Lewiston
Beaudet, Simon C.	25 Webster St., Lewiston
Beeaker, Vincent H.	85 Wood St., Lewiston
Beegel, Paul M.	80 Goff St., Auburn
Beliveau, Bertrand A.	56 Howe St., Lewiston
Branch, Charles F.	Central Maine Gen. Hosp., Lewiston
Brien, Maurice	76 Pine St., Lewiston
Busch, John J.	105 Elm St., Mechanic Falls
Carrier, John W.	Central Maine Gen. Hosp., Lewiston
Chapin, Milan A.	237 Turner St., Auburn
Clapp, Waldo A.	215 College St., Lewiston
Clapperton, Gilbert	300 Main St., Lewiston
Cloutier, Wilfrid A.	646 Main St., Lewiston
Cox, William V.	133 Court St., Auburn
DeCosta, Donald A.	Poland Spring
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Fishman, Louis N.	185 Webster St., Lewiston
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Friend, John W.	49 Hampton Ave., Auburn
Frost, Robert A.	93 Summer St., Auburn
Gauvreau, Norman O.	78 Pine St., Lewiston
Goldman, Morris E.	185 Webster St., Lewiston
Goldman, Richard N.	185 Webster St., Lewiston
Goodwin, Ralph A., Jr.	33 Court St., Auburn
Green, Ross W.	33 Court St., Auburn
Greene, John P.	19 Sabattus St., Lewiston
Greene, Merrill S. F.	466 Main St., Lewiston
Grimes, Gilbert R.	185 Webster St., Lewiston
Haas, Rudolph	480 Main St., Lewiston
Hannigan, Charles A.	85 Goff St., Auburn
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Harkins, Michael J.	437 Main St., Lewiston
Horsman, Donald H.	50 Goff St., Auburn
James, Chakmakis	47 Howe St., Lewiston
James, John A.	117 Goff St., Auburn
Konecki, John T.	St. Mary's Gen. Hosp., Lewiston
LaFlamme, Paul J.	106 Russell St., Lewiston
Leitman, Reuben	188 Sabattus St., Lewiston
Lichter, Horatio A.	54 Pine St., Lewiston
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Lynn, Geraldine	188 Russell St., Lewiston
Martel, Cyprien L., Jr.	91 Bartlett St., Lewiston
Mendes, Joseph M.	5 School St., Lisbon Falls
Mendros, John G.	111 Webster St., Lewiston
Milazzo, John	42 Elm St., Auburn
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Morin, Gerard L.	104 Ash St., Lewiston
Morissette, Russell A.	185 Webster St., Lewiston
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Nadeau, Lawrence A.	41 Sherbrooke Ave., Lewiston
O'Connell, George B.	11 Lisbon St., Lewiston
Potts, Ronald S.	Central Maine Gen. Hosp., Lewiston
Proulx, Harvey J.	92 Pine St., Lewiston
Rand, Carleton H.	219 Oak St., Lewiston
Rando, Joseph J.	111 Webster St., Lewiston
Reeves, Edward L.	179 Sabattus St., Lewiston
Reeves, Helene M.	179 Sabattus St., Lewiston

Rock, Daniel A.	477 Main St., Lewiston
Sanford, Theodore H.	117 Goff St., Auburn
Shems, Albert	487 Main St., Lewiston
Shields, Daniel R.	369 Main St., Lewiston
Spear, William	107 Main St., Lisbon Falls
Steele, Charles W.	472 Main St., Lewiston
Sweatt, Linwood A.	48 Drummond St., Auburn
Swett, Alfred E.	308 Minot Ave., Auburn
Tchao, Jou S.	82 Pine St., Lewiston
Thacher, Henry C.	117 Goff St., Auburn
Tibbetts, Otis B.	181 Gamage Ave., Auburn
Tousignant, Camille	111 Pine St., Lewiston
Turcotte, Richard W.	70 Pine St., Lewiston
Wakefield, Robert D.	St. Mary's Gen. Hospital, Lewiston
Webber, Wedgwood P.	299 Main St., Lewiston
Willie, James A.	111 Webster St., Lewiston
Young, E. Stanley	Poland Spring
Zanca, Ralph	86 Pine St., Lewiston

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Pratt, Harold S.	Livermore Falls
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Williams, James A.	40 Pleasant St., Mechanic Falls

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Giguere, Eustache N.	90 Webster St., Lewiston

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*Secretary-Treasurer* — Clyde I. Swett, M.D.

### ACTIVE MEMBERS

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Albert, Joseph L.	4 Pleasant St., Fort Kent
Aungst, Melvin R.	Morneault Bldg., Fort Kent
Berberian, George M.	12 Lincoln St., Van Buren
Boone, Storer W.	429 Main St., Presque Isle
Brennan, Thomas V.	P.O. Box 1026, Atascadero, Calif.
Burr, Charles G.	90 Court St., Houlton
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Collins, H. Douglas	Caribou Clinic, Caribou
Donahue, Clement L.	22 Sweden St., Caribou
Donahue, Gerald H.	4 Station St., Presque Isle
Dunham, Marguerite C.	P.O. Box 748, Presque Isle
Etscovitz, Eli A.	Cary Mem. Hosp., Caribou
Giberson, Raymond G.	156A Academy St., Presque Isle
Gormley, Eugene G.	Market Square, Houlton
Gregory, Frederick J.	So. Main St., Caribou
Griffiths, Eugene B.	429 Main St., Presque Isle
Harrison, George J.	Market Square, Houlton
Harvey, Thomas G.	59 Mayo St., Caribou
Hayward, I. Mead	So. Main St., Caribou
Helfrich, Harry M., Jr.	122 Academy St., Presque Isle
Higgins, George F.	122 Academy St., Presque Isle
Hogan, Chester F.	62 Main St., Houlton
Johnson, Gordon N.	P.O. Box 86, Houlton
Johnson, R. Paul	Main St., Fort Kent
Kimball, Herrick C.	P.O. Box 372, Fort Fairfield

Kirk, William V.  
Labbe, Onil B.  
Lagace, Alfred F.  
MacDonald, Lewis V. A.  
Madigan, John B.  
Nicholas, Eric F.  
O'Brien, William A.

Ouellette, Benoit  
Page, Rosario A.  
Pendleton, Arthur D.  
Pines, Philip  
Price, Richard D.  
Proctor, Ray A.  
Reynolds, Arthur P.  
Smith, Carroll H.  
Smith, Margaret S.  
Somerville, Robert B.  
Somerville, Wallace B.  
Swett, Clyde I.  
Toussaint, Leonid G.  
White, Leland M.  
Williams, Edward P.  
Wilson, G. Ivan

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Van Buren  
Cary Mem. Hosp., Caribou  
Main St., Washburn  
Houlton  
11 Green St., Fort Fairfield  
Arthur R. Gould Mem. Hosp.,  
Presque Isle  
77 Main St., Fort Kent  
22 Sweden St., Caribou  
3 Green St., Fort Fairfield  
Maine St., Limestone  
R.F.D. 2, Caribou  
Garden Circle, Caribou  
29 Second St., Presque Isle  
Box 785, Presque Isle  
Box 967, Presque Isle  
45 Hillside St., Presque Isle  
Mars Hill  
18 Sherman St., Island Falls  
P.O. Box 9, Fort Kent  
So. Main St., Caribou  
72 Main St., Houlton  
40 Court St., Houlton

Bidwell, Robinson L.  
Bischoffberger, John M.  
Bisgrove, John G.  
Bishop, Lloyd W.  
Blaisdell, Elton R.  
Blumberg, Edward  
Bonney, James H.  
Bove, Louis G.  
Bowman, Peter W.  
Branson, Sidney R.  
Broggi, Frank S.  
Brown, Douglas H.  
Burnett, Claude A., Jr.  
Burnham, Harold N.  
Burns, Robert M.  
Burrage, William C.  
Capron, Charles W.  
Carson, Robert S.  
Chatterjee, Manu  
Christensen, Harry E.  
Ciampi, Louis A.  
Clark, Frederick B.  
Clark, Richard I.  
Clarkin, Charles P.  
Cole, Donald P.  
Crane, Lawrence  
Cummings, George O., Jr.  
Cunningham, Alice N.  
D'Andrea, Anthony L.  
Daniels, Donald H.  
Davidson, David  
Davidson, Gisela K.  
Davies, Lloyd G.  
Derry, G. Hermann  
Dienst, Stanley G.  
Dionne, Maurice J.  
Doby, Tibor  
Dooley, Francis M.  
Dore, Kenneth E.  
Dorogi, Louis V.  
Doughinett, Otis J.  
Drake, Emerson H.  
Drexler, James E.  
Dunham, Carl E.  
Dyhrberg, Norman E.  
Earnhardt, Joseph B.  
Eppinger, Ernst  
Fagone, Francis A.  
Ferguson, Franklin F.  
Fife, James L.  
Finks, Henry B.  
Fish, Nicholas  
Fox, Francis H.  
Galen, Robert S.  
Gates, Clifford W.  
Geer, Charles R.  
Geer, George I., Jr.  
Getchell, Ralph A.  
Geyerhahn, George  
Gibbons, John F.  
Glassmire, Charles R.  
Godsoe, John A.  
Goduti, Richard J.  
Goldfarb, Jaime  
Good, Philip G.  
Greco, Edward A.  
Grish, Albert J.  
Hallett, George W., Jr.  
Hanley, Daniel F.  
Hawkes, Richard S.  
Hecht, Henry  
Heifetz, Ralph  
Herrick, Stanley E., Jr.  
Hiebert, Clement A.  
Hill, Douglas R.  
Hinckley, Harris  
Holt, C. Lawrence  
Hudson, Henry A.

31 Bramhall St., Portland  
Naples  
165 Park Row, Brunswick  
211 Vaughan St., Portland  
12 Deering St., Portland  
Box C, Pownal  
229 Vaughan St., Portland  
12 Deering St., Portland  
Box C, Pownal  
37 Main St., So. Windham  
18 Neal St., Portland  
548 Shore Rd., Cape Elizabeth  
59 Deering St., Portland  
130 Main St., Gorham  
P.O. Box 151, Westbrook  
57 Deering St., Portland  
22 Bramhall St., Portland  
11 McKeen St., Brunswick  
11 McKeen St., Brunswick  
So. Freeport  
Gray  
131 State St., Portland  
Freeport Medical Ctr., Freeport  
64 Brookside Rd., Portland  
45 Deering St., Portland  
157 Pine St., Portland  
47 Deering St., Portland  
32 Federal St., Brunswick  
131 State St., Portland  
R.R. No. 1, Readfield  
235 State St., Portland  
235 State St., Portland  
78 Main St., Fryeburg  
690 Congress St., Portland  
190 Pine St., Portland  
Baribeau Dr., Brunswick  
131 State St., Portland  
53 Deering St., Portland  
133 Main St., Fryeburg  
149 Main St., Freeport  
763 Congress St., Portland  
18 Bramhall St., Portland  
Ward Town Rd., Freeport  
188 State St., Portland  
323 Main St., Cumberland Mills  
55 Stroudwater St., Westbrook  
52 Belmont St., Portland  
312 Congress St., Portland  
22 Bramhall St., Portland  
Baribeau Dr., Brunswick  
73 Deering St., Portland  
235 State St., Portland  
83 West St., Portland  
6 Breckan St., Brunswick  
130 Main St., Gorham  
690 Congress St., Portland  
690 Congress St., Portland  
14 Elmwood Ave., Cape Elizabeth  
73 Deering St., Portland  
22 Bramhall St., Portland  
58 Deering St., Portland  
19 Deering St., Portland  
9 Deering St., Portland  
Box C, Pownal  
38 Deering St., Portland  
12 Pine St., Portland  
Box C, Pownal  
72 West St., Portland  
P.O. Box 637, Brunswick  
47 Deering St., Portland  
326 Stevens Ave., Portland  
173 State St., Portland  
12 Deering St., Portland  
18 Bramhall St., Portland  
855 Sawyer St., So. Portland  
331 Cottage Rd., So. Portland  
27 Deering St., Portland  
R.F.D. No. 1, West Bridgton

#### SENIOR MEMBERS

Brown, Stephen S. Mars Hill  
Faucher, Francois J. Grand Isle

#### AFFILIATE MEMBERS

Helfrich, Nancy R. 48 Third St., Presque Isle  
Osborne, John R. Narrows Pond Rd., Winthrop  
Savage, Richard L. 4 Elm St., Fort Kent

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Frenette, Francis F. 13479 Clifton Blvd., Lakewood 7, Ohio  
Herson, Joseph H. 343 E. 30th St., New York, N. Y., 10016  
Rideout, Samuel 72 Faire Harbour, New London, Conn.

#### SERVICE MEMBER

Harvey, William C. 59 Mayo St., Caribou

#### CUMBERLAND COUNTY

*President* — Charles R. Geer, M.D.

*Secretary-Treasurer* — Stanley B. Sylvester, M.D.

#### ACTIVE MEMBERS

Adams, Marvin C. 32 Deering St., Portland  
Agan, Robert W. 144 State St., Portland  
Allen, Donald E. Sebago Lake  
Analís, Harry 3 Forest Pk., Portland  
Andrews, Anneliese M. Maine Medical Ctr., Portland  
Ansell, Harvey B. 39 Deering St., Portland  
Applin, Hilton H. 6 Cumberland St., Brunswick  
Aranson, Albert 39 Deering St., Portland  
Asali, Louis A. 29 Deering St., Portland  
Asherman, Edward G. 131 Chadwick St., Portland  
Bacastow, Merle S. 22 Bramhall St., Portland  
Bachrach, Louis 16 Union St., Brunswick  
Baldini, Elio 22 Bramhall St., Portland  
Baldwin, Warren C. 42 Deering St., Portland  
Ballard, Michael D. 38 Deering St., Portland  
Barnes, Kirk K. 11 McKeen St., Brunswick  
Bearor, Robert A. Maine Medical Ctr., Portland  
Bennet, Eben T. 49 Deering St., Portland  
Bergmann, Jerome W. 255 Western Prom., Portland  
Bettle, Ronald A. 32 Federal St., Brunswick  
Betts, Anthony Maine Medical Ctr., Portland



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## FRANKLIN COUNTY

*President* — Stanley B. Covert, M.D.

*Secretary-Treasurer* — Maynard B. Colley, M.D.

### ACTIVE MEMBERS

Browne, Hays G.	9A Main St., Farmington
Brinkman, Harry	47 Perham St., Farmington
Brinkman, Paul A.	Farmington
Colley, Maynard B.	14 Main St., Farmington
Covert, Stanley B.	Kingfield
Duffy, Wallace H.	100 Main St., Farmington
Eastman, Charles W.	15 Millett St., Livermore Falls
Fiorica, Gaetano T.	12 Church St., Chisholm
Floyd, Paul E.	2 Middle St., Farmington
Marsters, David W.	Phillips
Pope, W. Dean	6 Pleasant St., Rangeley
Reed, James W.	18 Main St., Farmington
Rowe, Gunther H.	42 Main St., Livermore Falls
Zikel, Herbert M.	High St., Wilton

### HONORARY MEMBER

Pratt, George L. 7 Main St., Farmington

### SENIOR MEMBER

Weymouth, Currier C. Eastmont Sq., Farmington

## HANCOCK COUNTY

*President* — Elizabeth E. Williamson, M.D.

*Secretary-Treasurer* — Russell G. Williamson, M.D.

### ACTIVE MEMBERS

Bromley, William C.	Medical Center
	Herrick Rd., Southwest Harbor
Brownlow, Bradley E.	Blue Hill Mem. Hosp., Blue Hill
Cameron, Dwight	Rockend Rd., Northeast Harbor
Coffin, Ernest L.	Northeast Harbor
Cooper, Llewellyn W.	194 Main St., Bar Harbor
Crowe, James H.	57 Main St., Ellsworth
Cruikshank, Frank S., Jr.	22 Forest St., Bar Harbor
Gray, Philip L.	Blue Hill
Hsu, Theodore S.	14 High St., Ellsworth
Joost, Arthur M., Jr.	P.O. Box B, Bucksport
Knickerbocker, Charles H.	15 High St., Bar Harbor
Kopfmann, Harry	Deer Isle
Lambdin, Morris A.	Maine Coast Mem. Hosp., Ellsworth
LaCasce, Joseph H.	50 Union St., Ellsworth
Lane, Russell M.	Water St., Blue Hill
Larrabee, Charles F.	48 Mt. Desert St., Bar Harbor
McIntyre, John D.	50 Union St., Ellsworth
McLarn, William D.	50 Union St., Ellsworth
O'Meara, Edward S.	Maine Coast Mem. Hosp., Ellsworth
Russell, Robert F.	Penobscot
Spencer, Earle W.	Maine Coast Mem. Hosp., Ellsworth
Suyama, Eji	58 W. Main St., Ellsworth
Thegen, W. Edward	Elm St., Bucksport
Torrey, Marcus A.	75 State St., Ellsworth
Weymouth, Raymond E.	194 Main St., Bar Harbor
Wilbur, Herbert T., Jr.	100 Main St., Southwest Harbor
Williamson, Elizabeth E.	Blue Hill
Williamson, Russell G.	Blue Hill Mem. Hosp., Blue Hill

### SENIOR MEMBER

Babcock, Harold S. Castine

### SERVICE MEMBERS

Black, Paul E. (Capt.) MC USN First Marine Air Wing,  
Navy #955 — F.P.O., San Francisco, Calif.  
York, Elihu 2 E. Sunset Ave., Apt. 2,  
Warrington, Florida 32507

## KENNEBEC COUNTY

*President* — Kenneth W. Sewall, M.D.

*Secretary-Treasurer* — Earle M. Davis, M.D.

### ACTIVE MEMBERS

Ashley, Alta	11 Weston St., Augusta
Barnard, John M. H.	Doctors' Park, 89 Hospital St., Augusta
Barron, Richard E.	Main St., Monmouth
Bauman, Clair S.	159 Silver St., Waterville
Beckerman, Stanley C.	82 Elm St., Waterville
Bhatnagar, Hemendra N.	30 Elm St., Waterville
Bolduc, Jean L.	173 Main St., Waterville
Bourassa, Harvey J.	15 Silver St., Waterville
Boytar, Alexander	V. A. Hospital, East Orange, N. J.
Brann, Henry A.	31 Western Ave., Augusta
Bull, Frank B.	72 Church St., Gardiner
Canal, Ory D.	Augusta State Hosp., Augusta
Castellanos, Jose	Augusta State Hosp., Augusta
Chamberlin, Richard T.	14 Gilman St., Waterville
Chasse, Richard L.	18 Park St., Waterville
Chen, Jen-Ti	Cherry Hill Terrace, Waterville
Cook, Aaron	23 High St., Waterville
Crawford, Joseph R.	105 Water St., Augusta
Dachslager, Philip	21 Western Ave., Augusta
Darlington, Brinton T.	Doctors Park,
	89 Hospital St., Augusta
Davis, Earle M.	2 School St., Waterville
Denison, John D.	105 Brunswick Ave., Gardiner
Dennis, Richard H.	33 College Ave., Waterville
Dore, Clarence E.	2 School St., Waterville
Dunn, Robert H.	Veterans Adm., Togus
Emanuel, Meyer	Veterans Adm., Togus
English, Lena M.	489 Castle Shannon Blvd., Pittsburgh, Pa. 15234
Ervin, Edmund N.	2 School St., Waterville
Fisher, Dean H.	State House, Augusta
Fisher, Samson	173 Main St., Waterville
Giddings, Lane	6 E. Chestnut St., Augusta
Giddings, Paul D.	31 Western Ave., Augusta
Giesen, Joseph H.	34 Gilman St., Waterville
Giguere, Leandre	30 Elm St., Waterville
Gingras, Adolphe J.	105 Water St., Augusta
Gingras, Napoleon J.	6 E. Chestnut St., Augusta
Goodof, Irving I.	Thayer Hospital, Waterville
Goodrich, Blynn O.	165 Main St., Waterville
Gould, George I.	79 Main St., Richmond
Guillemette, Maurice R.	109 Water St., Augusta
Guite, L. Armand	45 Elm St., Waterville
Harlow, Edwin W.	177 Main St., Waterville
Hill, Howard F.	33 College Ave., Waterville
Hill, Kevin	33 College Ave., Waterville
Hirschberger, Celia	44 Main St., Waterville
Hornberger, H. Richard	2 School St., Waterville
Hurd, Allan C.	72 Church St., Gardiner
Jan, M. Rafiq	1065 Forest Dr., Ancora, N. J.
Jones, Paul A., Jr.	2 School St., Waterville
Landwehr, George R.	21 Western Ave., Augusta
Lansing, Peter F.	16 Macomber Ave., Augusta
Lepore, Anthony E.	72 Church St., Gardiner
Lopez, Edwardo A.	33 College Ave., Waterville
Marshall, Joseph A.	177 Main St., Waterville
Mathews, Hugh J., Jr.	345 Water St., Gardiner
McLaughlin, Clarence R.	345 Water St., Gardiner
McLaughlin, Ivan E.	345 Water St., Gardiner
McQuillan, Arthur H.	177 Main St., Waterville
Melendy, Oakley A.	Doctors Park, 89 Hospital St., Augusta
Milliken, Howard H.	105 Second St., Hallowell
Monsivais, Alfredo	Augusta State Hosp., Augusta
Moore, Valentine J.	Thayer Hospital, Waterville
Morris, Craig W.	50 Bangor St., Augusta
O'Connor, Francis J.	4 Woodlawn St., Augusta
Ohler, Robert L.	Veterans Adm., Togus
Papadopoulos, George	Box 724, State Hospital, Augusta
Patterson, John C.	Box 724, State Hospital, Augusta
Peddie, Harry M. K.	Doctors Park,
	89 Hospital St., Augusta



Pfeiffer, Paul H.	14 Gilman St., Waterville
Plimpton, Jay R.	283 Water St., Augusta
Pomerleau, Ovid F.	179 Main St., Waterville
Pomerleau, Rodolphe J. F.	27 Main St., Waterville
Poulin, Albert A.	Cherry Hill Dr., Waterville
Poulin, James E.	177 Main St., Waterville
Pratt, Loring W.	177 Main St., Waterville
Provost, Helen C.	48 Green St., Augusta
Provost, Pierre E.	48 Green St., Augusta
Reynolds, John F.	216 Main St., Waterville
Richards, Lee W., Jr.	21 Western Ave., Augusta
Robertson, George J.	171 Harrison Ave., Boston 11, Mass.
Rodriguez, Jose M.	109 Silver St., Waterville
Runyon, William N.	283 Water St., Augusta
Russell, Theodore M.	21 Western Ave., Augusta
Sanders, Stephen W.	120 Main St., Winthrop
Saunders, Allen I.	Ferry Rd., R.F.D. 2, Augusta
Schmidt, Lorrimer M.	Veterans Adm., Togus
Schumacher, William E.	14 Westwood Rd., MD "B," Augusta
Schwarz, Harald J.	Sisters Hospital, Waterville
Seligman, Morris J.	Veterans Adm., Togus
Senenky, Joseph P.	Augusta State Hosp., Augusta
Sewall, Kenneth W.	2 School St., Waterville
Shaw, John H.	131 Sewall St., Augusta
Shelton, M. Tieche	21 Western Ave., Augusta
Simpson, Margaret R.	Box 275, Togus
Smith, Kenneth E.	Veterans Adm., Togus
Sommerfeld, Kurt A.	Veterans Adm., Togus
Southern, Edward M.	34 Gilman St., Waterville
Spellman, Francis A.	Veterans Adm., Togus
Stinchfield, Allan J.	P.O. Box 343, Augusta
Stocks, Joseph F.	67 Silver St., Waterville
Sturtevant, Vaughn R.	33 College Ave., Waterville
Towne, Charles E.	18 Common St., Waterville
Trembly, Bruce	33 College Ave., Waterville
Veilleux, Lucien F.	30 Elm St., Waterville
Weltman, Joseph S.	Veterans Adm., Togus
Willard, Harold N.	Thayer Hospital, Waterville
Wilson, Robert W.	Veterans Adm., Togus

#### HONORARY MEMBERS

Crawford, J. Ramser	105 Water St., Augusta
Hill, Frederick T.	Thayer Hospital, Waterville
Kagan, Samuel H.	283 Water St., Augusta
McKay, Roland L.	P.O. Box 265, Augusta
Newcomb, Charles H.	Clinton
Reynolds, Ralph L.	216 Main St., Waterville
Shannon, Charles E. G.	9 Park St., Waterville

#### SENIOR MEMBERS

Breard, Alfred	15 Summer St., Waterville
Crawford, Albert S.	East Blue Hill
Herring, Leon D.	Memorial Dr., Winthrop
Langer, Ella	State House, Augusta
Marquardt, Matthias	Augusta State Hosp., Augusta

#### AFFILIATE MEMBERS

Reel, John J.	59 So. Front St., Richmond
Sleeper, Francis H.	19 Columbia St., Augusta
Tashiro, Sabro	181 Highland Ave., Gardiner

#### JUNIOR MEMBER

Michaud, Joseph C.	6377 Eldredge Rd., Bedford Heights, Ohio
--------------------	---

#### KNOX COUNTY

*President* — John A. Root, M.D.

*Secretary-Treasurer* — Onni C. Kangas, M.D.

#### ACTIVE MEMBERS

Alexander, Fay K.	Camden Community Hosp., Camden
Alfaro, Ciro	Atlantic Ave. and Sea St., Camden

Apollonio, Howard L.
Brouwer, Johan
Dennison, Frederick C.
Earle, Ralph P.
Eddy, Robert H.
Fuller, Barbara L.
Hawkins, Donald B.
Heath, Parker, Jr.
Holz, Peter H.
Hopping, John S.
Howard, Emery B.
Hunter, Albert L.
Jones, Paul A., Sr.
Kangas, Onni C.
Kibbe, Frank W.
King, Merrill J., Sr.
King, Merrill J., Jr.
Lawry, Oram R., Jr.
Mann, David V.
McLellan, William A.
Millington, Paul A.
Morse, Edward K.
Onat, Mustafa V.
Root, John A.
Soule, Gilmore W.
Tounge, Harry G., Jr.
Ward, William W.
Wagatt, Wesley N.
Waterman, Richard
White, Henry O.
Worthing, Verla E.

22 White St., Rockland
5 Beech St., Rockland
183 Main St., Thomaston
Vinalhaven
5 Beech St., Rockland
20 Chestnut St., Rockland
Atlantic Ave. & Sea St., Camden
22 White St., Rockland
51 Elm St., Camden
R.F.D. No. 2, Union
23A Summer St., Rockland
Knox County Gen. Hosp., Rockland
Union
417 Main St., Rockland
R.F.D., Lincolnville
9 Deering St., Portland
22 White St., Rockland
96 Limerock St., Rockland
22 White St., Rockland
87 Chestnut St., Camden
44 Mountain St., Camden
22 White St., Rockland
St. George
22 White St., Rockland
22 White St., Rockland
12 Union St., Camden
P.O. Box 804, Rockland
41 Talbot Ave., Rockland
Main St., Waldoboro
22 White St., Rockland
Box A, Thomaston

#### HONORARY MEMBERS

Campbell, Fred G.	P.O. Box 484, Warren
Hall, Walter D.	407 Main St., Rockland

#### SENIOR MEMBERS

Frost, Harold M.	Friendship
Hochschild, Hugo	33 Main St., Thomaston
Jameson, C. Harold	Medical Arts Bldg., Rockland
Loewenstein, George	Chebeague Island
	Winter address — Aripeka, Florida
Platt, Anna	Beauchamp Rd., Rockport
	Winter — 110 Manatee Rd., Belleair, Clearwater, Fla.

#### AFFILIATE MEMBERS

Bean, Achsa M.	Star Route 22-282, Owl's Head
Stimson, Barbara B.	Star Route 22-282, Owl's Head
Waterman, Dorothy	Waldoboro

#### LINCOLN-SAGADAHOC COUNTY

*President* — Edward L. Kinder, Jr., M.D.

*Secretary-Treasurer* — George W. Bostwick, M.D.

#### ACTIVE MEMBERS

Akar, Hamdi	17 Grove St., Bath
Andrews, John F.	20 West St., Boothbay Harbor
Belknap, Samuel L.	Damariscotta
Blackburn, Nelson P.	Bath Memorial Hosp., Bath
Bostwick, George W.	Newcastle
Burden, Charles E.	1 North St., Bath
Doble, Miriam	990 Washington St., Bath
Dougherty, John F.	112 Front St., Bath
Fichtner, Paul A.	781 High St., Bath
Gregory, Philip O.	St. Andrews Hosp., Boothbay Harbor
Griffin, Carl R., Jr.	69 Townsend Ave., Boothbay Harbor
Kinder, Edward L., Jr.	1027 Washington St., Bath
Lenfest, Stanley R.	Waldoboro
McLaren, John J.	Baribeau Dr., Brunswick
Nichols, Arthur A.	Edgecomb

Oceretko, Arkadij	37 Court St., Bath
Powell, Ralph C.	Damariscotta
Proctor, Thomas E.	Boothbay Harbor
Smith, Jacob	118 Front St., Bath
Smith, Joseph I.	118 Front St., Bath
Stetkevych, Alexander G.	858 Washington St., Bath
Tracy, Mary J.	Bristol Rd., Damariscotta
Wilson, Harry M.	944 Middle St., Bath
Winchenbach, Francis A.	910 Washington St., Bath
Zeller, Alan W.	35 Main St., Damariscotta

#### HONORARY MEMBERS

Barrows, Harris C.	115 Cony St., Augusta
Kershner, Warren E.	57 Green St., Bath
Morin, Harry F.	905 Middle St., Bath
Stetson, Rufus E.	Damariscotta

#### SENIOR MEMBER

Dalrymple, Sidney C.	So. Great Rd., So. Lincoln, Mass.
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#### AFFILIATE MEMBERS

Bachus, John M.	1023 High St., Bath
Hamilton, Virginia C.	So. Harpswell
Sherman, Fuller G.	Spruce Pt., Boothbay Harbor

#### OXFORD COUNTY

*President* — Leonidas B. Kudisch, M.D.

*Secretary-Treasurer* — Albert P. Royal, Jr., M.D.

#### ACTIVE MEMBERS

Akerberg, Ake	2 East Main St., So. Paris
Aucoin, Peter B.	151 Franklin St., Rumford
Bean, H. Richard	241 Main St., Norway
Broughton, David S.	18 Hartford St., Rumford
Defoe, Garfield G.	Dixfield
Dixon, Walter G.	16 Deering St., Norway
Elsemore, Dexter E.	11 Main St., Dixfield
Harper, Harry L.	17 Main St., So. Paris
Hiebert, Joelle, C., Jr.	Box 148, Norway
Howard, Henry M.	105 Franklin St., Rumford
Jackson, Norman M.	151 Franklin St., Rumford
Kudisch, Leonidas B.	11 Franklin St., Rumford
Lord, Edwin M.	39 Franklin St., Rumford
Martin, Joseph E.	35 Main St., Mexico
McCormack, Roland L.	12 Bridge St., Norway
Moore, Beryl M.	Oxford
Nangle, Thomas P.	West Paris
Oestrich, Alfred	89 Congress St., Rumford
Royal, Albert P., Jr.	82 Maine Ave., Rumford
Smith, Charles M.	Main St., Dixfield
Young, John	Paradise St., Bethel

#### HONORARY MEMBERS

Adams, Lester	9 Knox St., Thomaston
Mills, Nathaniel	North Main St., Wolfeboro, N. H.
Pearson, Henry	Brownfield
Stanwood, Harold W.	Dixfield

#### SENIOR MEMBERS

Hubbard, Roswell E.	Waterford
Kay, Edwin	31 Frye St., Lewiston
MacDougall, James A.	303 Penobscot St., Rumford
Nelson, Chesley W.	121 Main St., Norway

#### JUNIOR MEMBER

Rowe, Linwood M.	22 Bramhall St., Portland
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#### SERVICE MEMBER

Boynton, Willard H.	U.S. A.I.D., A.P.O. 271, New York, N. Y.
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#### PENOBSCOT COUNTY

*President* — William A. Purinton, M.D.

*Secretary* — Hadley Parrot, M.D.

*Treasurer* — Benjamin L. Shapero, M.D.

#### ACTIVE MEMBERS

Adams, Asa C.	68 Main St., Orono
Adams, Winford C.	255 N. Main St., Brewer
Babcock, Edward B.	316 State St., Bangor
Barrett, Robert J., Jr.	Cor. Union & James Sts., Bangor
Bjorn, John C.	Hampden Highlands
Braisden, Carl E.	47 Broadway, Bangor
Blaisdell, William B., Jr.	47 Broadway, Bangor
Blinder, Philip	128 Broadway, Bangor
Bridges, Donald E.	336 Mt. Hope Ave., Bangor
Brod, James J.	51 Grove St., Bangor
Brown, Eugene E.	57 Summit Ave., Bangor
Brown, Lloyd	186 State St., Bangor
Burke, John E.	824 State St., Bangor
Burke, Paul W.	5 High St., Newport
Butler, Harry	77 Broadway, Bangor
Butterfield, Wilfred I.	119 Main St., Lincoln
Chase, George O.	Eastern Maine Gen. Hosp., Bangor
Chason, Sidney	128 Broadway, Bangor
Clement, James D., Jr.	77 Essex St., Bangor
Clough, Dexter J., 2nd	224 State St., Bangor
Cornell, Robert C.	118 Forest Ave., Orono
Coulton, Donald	326 State St., Bangor
Cross, Harold D.	Main Rd. & Summer Sts., Hampden Highlands
Curran, Edward L.	209 State St., Bangor
Cutler, Lawrence M.	31 Grove St., Bangor
Desjardins, Richard F.	240 Penobscot Ave., Millinocket
Dietrich, Mary M.	P.O. Box 93, Orrington
Duffey, Richard V.	187 N. Main St., Brewer
Dwyer, Clement S.	205 French St., Bangor
Emery, Frederick C.	242 Cedar St., Bangor
Eyerer, Rudolf E.	489 State St., Bangor
Feeley, J. Robert	438 Garland St., Bangor
Fergus, Andrew	128 Broadway, Bangor
Gaillard, Richard A.	276 State St., Bangor
Gilman, Herbert C.	240 Penobscot Ave., Millinocket
Graves, Robert A.	Sunset Drive, Orono
Hall, Walter L. H.	130 Middle St., Old Town
Hamlin, Irving E.	Main St., E. Millinocket
Hill, Allison K.	113 Somerset St., Bangor
Houlihan, John S.	209 State St., Bangor
Hughes, Edward J., Jr.	336 Mt. Hope Ave., Bangor
Hutchins, Deane L.	Health Dept., Univ. of Maine, Orono
Irwin, Carl W.	262 State St., Bangor
Kadi, Francis J.	Bangor State Hosp., Bangor
Kellogg, Robert O.	316 State St., Bangor
Leddy, Percy A.	Main St., Seal Harbor
Lee, Kong	22 Glenn Dr., Woodbury, L. I., N. Y.
Lieberman, Arthur N.	180 Broadway, Bangor
Macdonald, Donald F.	263 State St., Bangor
Manter, Wilbur B.	1 Fern St., Bangor
Mason, Peter H.	Millinocket Com. Hosp., Millinocket
McEvoy, Charles D., Jr.	186 State St., Bangor
McGinn, John F.	205 French St., Bangor
McLean, Preston A.	336 Mt. Hope Ave., Bangor
McNamara, Wesley C.	8 Lee St., Lincoln
McQuoid, Robert M.	39 Columbia St., Bangor
Memmelaar, Joseph E.	54 Forest Ave., Bangor
Merriam, Thornton W., Jr.	44 James St., Bangor
Miragliuolo, Leonard G.	10 Maple St., Bangor
Moulton, Gardner N.	5 Grove St., Bangor
Munce, Richard T.	262 State St., Bangor



Nesin, Bourcard	10 Water St., Howland
Netland, Anders T.	317 State St., Bangor
O'Kane, Francis R.	122 Penobscot Ave., Millinocket
Osler, Jay K.	74 Birch St., Bangor
Palmer, Thomas H., Jr.	316 State St., Bangor
Parrot, Hadley	74 Somerset St., Bangor
Pearson, John J.	100 S. Main St., Old Town
Pooler, Harold A.	Bangor State Hosp., Bangor
Porter, Edward C.	489 State St., Bangor
Purinton, William A.	276 State St., Bangor
Ridlon, Magnus F.	99 Broadway, Bangor
Rublin, Carl W.	205 French St., Bangor
Sewall, Elmer M.	14 Park St., Orono
Shapero, Benjamin L.	142 Pine St., Bangor
Shubert, Alice J.	317 State St., Bangor
Shubert, William M.	317 State St., Bangor
Shurman, Hans	10 Spring St., Dexter
Smith, Hugh A.	Eastern Maine Gen. Hosp., Bangor
Striar, Ronald R.	94 Essex St., Bangor
Strout, Warren G.	205 French St., Bangor
Sullivan, John R.	340 No. Main St., Brewer
Taylor, H. Lewis	25 Church St., Dexter
Thomas, Philip B.	205 French St., Bangor
Todd, Albert C.	185 No. Main St., Brewer
Trowbridge, Mason, Jr.	142 Pine St., Bangor
Vickers, Martyn A.	268 State St., Bangor
Wadsworth, Richard C.	489 State St., Bangor
Wagner, Samuel L.	2 Holmes St., Winterport
Walker, George R.	336 Mt. Hope Ave., Bangor
Warren, H. Draper	Eastern Maine Gen. Hosp., Bangor
Weisz, Hans	194 Main St., Lincoln
Whitney, Byron V.	280 State St., Bangor
Whitworth, John E.	116 Hammond St., Bangor
Wood, George W., III	156 No. Main St., Brewer
Woodcock, John A.	35 Second St., Bangor

**HONORARY MEMBERS**

Craig, Allan	905 Egan Ave., Pacific Grove, Calif.
Devan, Thomas A.	10245-47th Ave., Corona, L. I., N. Y.
Hedin, Carl J.	Penobscot Terrace, Brewer
Higgins, George I.	15 Water St., Newport
McNeil, Harry D.	81 Silver Rd., Bangor
Scribner, Herbert C.	200 Union St., Bangor
Weatherbee, George B.	Main St., Hampden Highlands

**SENIOR MEMBERS**

Ames, Forrest B.	255 Hammond St., Bangor
Dunham, Rand A.	Box 400, East Millinocket
Emerson, W. Merritt	131 State St., Bangor
Woodcock, Allan	35 Second St., Bangor

**AFFILIATE MEMBERS**

DeWitt, James C.	(Address Unknown)
Merrill, Urban H.	Etna

**SERVICE MEMBER**

Clough, Herbert T.	(Col.) Hq. USAF (AFCSG 12), Bldg. T-8, Washington 25, D. C.
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**PISCATAQUIS COUNTY**

*President* — Linus J. Stitham, M.D.

*Secretary-Treasurer* — Odd S. Nielson, M.D.

**ACTIVE MEMBERS**

Bradbury, Francis W.	16 E. Main St., Dover-Foxcroft
Carde, Albert M.	33 Elm St., Milo
Curtis, John B.	10 High St., Milo
Garcia-Rey, Felix M.	14 Charles St., Milo

Howard, George C.	Oak St., Guilford
Metcalf, John T.	47 Elm St., Milo
Lightbody, Charles H.	No. Main St., Guilford
Nelson, Isaac	Box 336, Greenville
Nielsen, Odd S.	85 Pleasant St., Dexter
Rodriguez, Araminta	14 Charles St., Milo
Stitham, Linus J.	50 Main St., Dover-Foxcroft
Stuart, Ralph C.	Guilford

**HONORARY MEMBERS**

Bundy, Harvey C.	Milo
MacDougal, Wilbur E.	186 Nowell Rd., Bangor
Pritham, Fred J.	Greenville Jct.

**SENIOR MEMBERS**

Nickerson, Norman H.	Greenville
Stanhope, Charles N.	South St., Dover-Foxcroft

**JUNIOR MEMBER**

Johnson, James H., Jr.	Pine St., Centerville, Mass.
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**SOMERSET COUNTY**

*President* — W. Edward Jordan, Jr., M.D.

*Secretary-Treasurer* — Marian L. Strickland, M.D.

**ACTIVE MEMBERS**

Amrein, H. Carl	29 Weston Ave., Madison
Ball, Franklin P.	Bingham
Bernard, Albert J.	198 Madison Ave., Skowhegan
Briggs, Paul R.	Hartland
Greenlaw, William A.	129 Main St., Fairfield
Grow, William B.	Central Maine San., Fairfield
Hoch, Gretl J.	Box 146, Jackman
Hornstein, Louis S.	220 Water St., Skowhegan
Jordan, W. Edward, Jr.	68 Water St., Skowhegan
Kemezys, Kestutis M.	25 Garfield St., Madison
Laney, Richard P.	50 Water St., Skowhegan
McIntire, Percy	Central Maine San., Fairfield
Philbrick, Maurice S.	292 Water St., Skowhegan
Reed, Howard L.	68 Water St., Skowhegan
Smith, Edgar J.	1 Park St., Fairfield
Soroka, Selic	39 High St., Skowhegan
Strickland, Marian L.	Easy St., Canaan
Sullivan, George E.	R.F.D. #1, Fairfield
Sy, Vincente L.	Milford Ave., Bingham
Szelenyi, Ernest	Central Maine San., Fairfield
Szendey, Andrew M.	26 Gray St., Madison
Turner, Harland G.	Box 38, Norridgewock

**HONORARY MEMBERS**

Humphreys, Ernest D.	91 Main St., Pittsfield
Marston, Henry E.	No. Anson
Webber, Merlon A.	33 Lancey St., Pittsfield

**SENIOR MEMBERS**

Lord, Maurice E.	Box 537, Lake Placid, Florida
Southworth, John D.	Hartland

**WALDO COUNTY**

*President* — Ernest W. Stein, M.D.

*Secretary-Treasurer* — Seth H. Read, M.D.

**ACTIVE MEMBERS**

Caswell, John A.	16 Waldo Ave., Belfast
Cobb, Norman E.	132 Main St., Belfast

Read, Seth H.  
 Stein, Ernest W.  
 Temple, George L.  
 Torrey, Raymond L.  
 Webber, John R.

15 Church St., Belfast  
 72 Main St., Pittsfield  
 Fahey St., Belfast  
 Main St., Searsport  
 6 Northport Ave., Belfast

#### HONORARY MEMBERS

Small, Foster C.  
 Stevens, Carl H.

169 High St., Belfast  
 18 Franklin St., Belfast

#### WASHINGTON COUNTY

*President* — James C. Bates, M.D.

*Secretary-Treasurer* — Karl V. Larson, M.D.

#### ACTIVE MEMBERS

Bates, James C.  
 Brownrigg, Leslie W.  
 French, Rowland B.  
 Jacob, Donald R.  
 Kazutow, John  
 Larson, Karl V.  
 MacBride, Robert G.  
 McAllister, John W.  
 Mitchell, Hazen C.  
 Mundie, Perley J.  
 Nackley, George N.  
 Rice, William C.  
 Sears, Harold G.  
 Shaw, George B.

Eastport  
 St. Stephen, N. B.  
 16 Water St., Eastport  
 Princeton  
 Box 113, Columbia Falls  
 E. Machias  
 25 Washington St., Lubec  
 39 Water St., Lubec  
 Calais  
 32 North St., Calais  
 1 School St., Machias  
 Main St., Calais  
 Second Ave., Woodland  
 Main St., Jonesport

#### SENIOR MEMBERS

Armstrong, Charles M.  
 Bennet, DaCosta F.

Robbinston  
 4 Main St., Lubec

#### YORK COUNTY

*President* — Roger J. P. Robert, M.D.

*Secretary-Treasurer* — Charles W. Kinghorn, M.D.

#### ACTIVE MEMBERS

Anton, Thomas  
 Bacon, Melvin  
 Belmont, Ralph S.  
 Binette, Germain A.  
 Charest, Leandre R.  
 Cuneo, Kenneth J.  
 Dionne, William E.  
 Downing, J. Robert  
 Drummond, S. Dunton  
 Endicott, Ruth E.

260 Main St., Biddeford  
 122 Main St., Sanford  
 6 Washington St., Sanford  
 331 Main St., Saco  
 314 Alfred St., Biddeford  
 31 Summer St., Kennebunk  
 75 Main St., Springvale  
 35 Summer St., Kennebunk  
 Bar Mills  
 16 Main St., Ogunquit

Ficker, Robert F.  
 Fortier, Andre P.  
 Haas, Carl M.  
 Hazzard, Lawrence R.  
 Hill, Paul S., Jr.  
 Hoffman, Alvin A.  
 Hopkins, Herbert J.  
 Houle, Marcel P.  
 Jellerson, Leon R.  
 Johnston, James S.  
 LaFond, Robert S.  
 Lapirow, Harry  
 Leigh, Kenneth E.  
 Lesieur, Louis C.  
 Lincourt, Armand S.  
 Lord, George A.  
 Lorentz, John J.  
 Magaudda, Michael M. P.

Magocsi, Alexander W.  
 Mahaney, William F.  
 Moulton, Marion K.  
 Murphy, John J.  
 Myer, John C.  
 O'Sullivan, William B.  
 Ouellette, Marcel D.  
 Patane, Joseph M.  
 Perrault, Oscar W.  
 Peterlein, Walter R., Jr.  
 Renzulli, Ottone  
 Richards, Carl E.  
 Robert, Roger J. P.  
 Ross, Maurice  
 Roussin, William T.  
 Shaw, G. Patrick  
 Smith, Gerald R.  
 Smith, Oney P.  
 Taylor, Paul E.  
 Turville, Charles S.  
 Vachon, Robert D.  
 Viger, Leopold A.  
 Wolfahrt, Eugene P.

Maine St., Kennebunkport  
 68 Foss St., Biddeford  
 357 Elm St., Biddeford  
 Breakfast Hill Rd., Greenland, N. H.  
 323 Main St., Saco  
 P.O. Box 222, York  
 24 Portland Ave., Old Orchard  
 200 Alfred St., Biddeford  
 77 Franklin St., Boston 12, Mass.  
 York Harbor  
 258 Main St., Saco  
 99 Main St., Kennebunk  
 Brixham Rd., York  
 255 Beach St., Saco  
 122 Main St., Sanford  
 34 Winter St., Sanford  
 Hyde Rehabilitation Ctr., Bath  
 39 Old Orchard St.,  
 Old Orchard Beach  
 York  
 338 Main St., Saco  
 W. Newfield  
 84 Portland St., So. Berwick  
 Nasson College, Springvale  
 331 Main St., Saco  
 114 Main St., Sanford  
 256 Alfred St., Biddeford  
 30 South St., Biddeford  
 75 Main St., Springvale  
 346 Elm St., Biddeford  
 34 Winter St., Sanford  
 331 Main St., Saco  
 372 Main St., Saco  
 48 Bacon St., Biddeford  
 357 Elm St., Biddeford  
 Ogunquit  
 Post Rd., Wells  
 9 Wentworth St., Kittery  
 P.O. Box 187, Alfred  
 34 Winter St., Sanford  
 176 Elm St., Biddeford  
 338 Main St., Saco

#### HONORARY MEMBERS

Bunker, Willard H.  
 Cobb, Stephen A.  
 Davis, Ansel S.  
 Larochelle, Joseph R.  
 Ross, H. Danforth  
 Sever, James W.  
 Whitney, Ray L.

York Harbor  
 34 Winter St., Sanford  
 Springvale  
 42 Bacon St., Biddeford  
 34 Winter St., Sanford  
 1800 Massachusetts Ave., Cambridge, Mass.  
 Cape Porpoise

#### SENIOR MEMBERS

Dennett, Carl G.  
 Kinghorn, Charles W.

258 Main St., Saco  
 4 Wentworth St., Kittery

#### JUNIOR MEMBER

Berger, Steven

Station B, Poughkeepsie, N. Y.



# An Alphabetical List of the Members of the Maine Medical Association

The figures in parentheses refer to County Societies as follows: (1) Androscoggin, (2) Aroostook, (3) Cumberland, (4) Franklin, (5) Hancock, (6) Kennebec, (7) Knox, (8) Lincoln-Sagadahoc, (9) Oxford, (10) Penobscot, (11) Piscataquis, (12) Somerset, (13) Waldo, (14) Washington, (15) York.

## A

Adams, Asa C., 68 Main St., Orono (10)  
 Adams, Lester, 9 Knox St., Thomaston (9)  
 Adams, Marvin C., 32 Deering St., Portland (3)  
 Adams, Winford C., 255 North Main St., Brewer (10)  
 Agan, Robert W., 144 State St., Portland (3)  
 Akar, Hamdi, 17 Grove St., Bath (8)  
 Akerberg, Ake, 2 East Main St., South Paris (9)  
 Albert, Armand, 193 Main St., Van Buren (2)  
 Albert, Joseph L., 4 Pleasant St., Fort Kent (2)  
 Alexander, Fay K., Camden Community Hosp., Camden (7)  
 Alfaro, Ciro, Atlantic Ave. and Sea St., Camden (7)  
 Allen, Donald E., Sebago Lake (3)  
 Ames, Forrest B., 255 Hammond St., Bangor (10)  
 Amfilo, Basil, 626 Main St., Lewiston (1)  
 Amrein, H. Carl, 29 Weston Ave., Madison (12)  
 Anais, Harry, 3 Forest Pk., Portland (3)  
 Anderson, Donald L., 369 Main St., Lewiston (1)  
 Anderson, Dorothy, 369 Main St., Lewiston (1)  
 Andrews, Anneliese M., Maine Medical Center, Portland (3)  
 Andrews, John F., 20 West St., Boothbay Harbor (8)  
 Ansell, Harvey B., 39 Deering St., Portland (3)  
 Anton, Thomas, 260 Main St., Biddeford (15)  
 Apollonio, Howard L., 22 White St., Rockland (7)  
 Applin, Hilton H., 6 Cumberland St., Brunswick (3)  
 Aranson, Albert, 39 Deering St., Portland (3)  
 Archambault, Philip L., 346 Main St., Lewiston (1)  
 Armstrong, Charles M., Robbinston (14)  
 Asali, Louis A., 29 Deering St., Portland (3)  
 Asherman, Edward G., 131 Chadwick St., Portland (3)  
 Ashley, Alta, 11 Weston St., Augusta (6)  
 Aucoin, Peter B., 151 Franklin St., Rumford (9)  
 Aungst, Melvin R., Morneau Building, Fort Kent (2)

## B

Babalian, Leon, 645 Congress St., Portland (3)  
 Babcock, Edward B., 316 State St., Bangor (10)  
 Babcock, Harold S., Castine (5)  
 Bacastow, Merle S., 22 Bramhall St., Portland (3)  
 Bachrach, Louis, 16 Union St., Brunswick (3)  
 Bachulus, John M., 1023 High St., Bath (8)  
 Bacon, Melvin, 122 Main St., Sanford (15)  
 Baldini, Elio, 22 Bramhall St., Portland (3)  
 Baldwin, Warren C., 42 Deering St., Portland (3)  
 Ball, Franklin P., Bingham (12)  
 Ballard, Michael D., 38 Deering St., Portland (3)  
 Barker, Nathaniel B. T., 1 South St., Yarmouth (3)  
 Barnard, John M. H., Doctors Park, 89 Hospital St., Augusta (6)  
 Barnes, Kirk K., 11 McKean St., Brunswick (3)  
 Barrett, Robert J., Jr., Cor. Union & James Sts., Bangor (10)  
 Barron, Richard E., Main St., Monmouth (6)  
 Barrows, Harris C., 115 Cony St., Augusta (8)  
 Bates, James C., Eastport (14)  
 Bauman, Clair S., 159 Silver St., Waterville (6)  
 Bean, Achsa M., Star Route 22-282, Owl's Head (7)  
 Bean, H. Richard, 241 Main St., Norway (9)  
 Bearor, Robert A., Maine Medical Ctr., Portland (3)  
 Beaudet, Simon C., 25 Webster St., Lewiston (1)  
 Beckerman, Stanley C., 82 Elm St., Waterville (6)  
 Beeaker, Vincent H., 85 Wood St., Lewiston (1)  
 Beegel, Paul M., 80 Goff St., Auburn (1)  
 Beliveau, Bertrand A., 56 Howe St., Lewiston (1)  
 Belknap, Samuel L., Damariscotta (8)  
 Belmont, Ralph S., 6 Washington St., Sanford (15)  
 Bennet, DaCosta F., 4 Main St., Lubec (14)  
 Bennet, Eben T., 49 Deering St., Portland (3)  
 Berberian, George M., 12 Lincoln St., Van Buren (2)

Berger, Steven, Station B, Poughkeepsie, N. Y. (15)  
 Bergmann, Jerome W., 255 Western Prom., Portland (3)  
 Bernard, Albert J., 198 Madison Ave., Skowhegan (12)  
 Bettle, Ronald A., 32 Federal St., Brunswick (3)  
 Betts, Anthony, Maine Medical Ctr., Portland (3)  
 Bhatnagar, Hemendra N., 30 Elm St., Waterville (6)  
 Bidwell, Robinson L., 31 Bramhall St., Portland (3)  
 Binette, Germain A., 331 Main St., Saco (15)  
 Bischoffberger, John M., Naples (3)  
 Bisgrove, John G., 165 Park Row, Brunswick (3)  
 Bishop, Lloyd W., 211 Vaughan St., Portland (3)  
 Bjorn, John C., Hampden Highlands (10)  
 Black, Paul E., Capt., First Marine Air Wing,  
 Navy #955-F.P.O., San Francisco, Calif. (5)  
 Blackburn, Nelson P., Bath Memorial Hosp., Bath (8)  
 Blaisdell, Carl E., 47 Broadway, Bangor (10)  
 Blaisdell, Elton R., 12 Deering St., Portland (3)  
 Blaisdell, William B., Jr., 47 Broadway, Bangor (10)  
 Blinder, Philip, 128 Broadway, Bangor (10)  
 Blumberg, Edward, Box C, Pownal (3)  
 Bolduc, Jean L., 173 Main St., Waterville (6)  
 Bonney, James H., 229 Vaughan St., Portland (3)  
 Boone, Storer W., 429 Main St., Presque Isle (2)  
 Bostwick, George W., Newcastle (8)  
 Bourassa, Harvey J., 15 Silver St., Waterville (6)  
 Bove, Louis G., 12 Deering St., Portland (3)  
 Bowman, Peter W., P.O. Box C, Pownal (3)  
 Bowne, Hays G., 9A Main St., Farmington (4)  
 Boynton, Willard H., U.S. A.I.D., A.P.O. 271,  
 New York, N. Y. (9)  
 Boytar, Alexander A., V. A. Hospital, East Orange, N. J. (6)  
 Bradbury, Francis W., 16 E. Main St., Dover-Foxcroft (11)  
 Bramhall, Theodore C., 185 Craigie St., Portland (3)  
 Winter address—3531 Mineola Dr., Sarasota, Fla.  
 Branch, Charles F., Central Maine Gen. Hosp., Lewiston (1)  
 Brann, Henry A., 31 Western Ave., Augusta (6)  
 Branson, Sidney R., 37 Main St., South Windham (3)  
 Breard, J. Alfred, 15 Summer St., Waterville (6)  
 Brennan, Thomas V., P.O. Box 1026, Atascadero, Calif. (2)  
 Bridges, Donald E., 336 Mt. Hope Ave., Bangor (10)  
 Brien, Maurice, 76 Pine St., Lewiston (1)  
 Briggs, Paul R., Hartland (12)  
 Brinkman, Harry, 47 Perham St., Farmington (4)  
 Brinkman, Paul A., Farmington (4)  
 Brod, James J., 51 Grove St., Bangor (10)  
 Broggi, Frank S., 18 Neal St., Portland (3)  
 Bromley, William C., Medical Ctr., Herrick Rd.,  
 Southwest Harbor (5)  
 Broughton, David S., 18 Hartford St., Rumford (9)  
 Brouwer, Jehan, 5 Beech St., Rockland (7)  
 Brown, Douglas H., 548 Shore Rd., Cape Elizabeth (3)  
 Brown, Eugene E., 57 Summit Ave., Bangor (10)  
 Brown, Lloyd, 186 State St., Bangor (10)  
 Brown, Luther A., Eastland Motor Hotel, Portland (3)  
 Brown, Stephen S., Mars Hill (2)  
 Brownlow, Bradley E., Blue Hill Mem. Hosp., Blue Hill (5)  
 Brownrigg, Leslie W., St. Stephen, N. B. (14)  
 Buker, Edson B., R.F.D. No. 3, Auburn (1)  
 Bull, Frank B., 72 Church St., Gardiner (6)  
 Bundy, Harvey C., Milo (11)  
 Bunker, Willard H., York Harbor (15)  
 Burden, Charles E., 1 North St., Bath (8)  
 Burke, John E., 824 State St., Bangor (10)  
 Burke, Paul W., 5 High St., Newport (10)  
 Burnett, Claude A., Jr., 59 Deering St., Portland (3)  
 Burnham, Harold N., 130 Main St., Gorham (3)  
 Burns, Robert M., P.O. Box 151, Westbrook (3)  
 Burr, Charles G., 90 Court St., Houlton (2)  
 Burrage, William C., 57 Deering St., Portland (3)  
 Busch, John J., 105 Elm St., Mechanic Falls (1)  
 Butler, Harry, 77 Broadway, Bangor (10)  
 Butterfield, Wilfred L., 119 Main St., Lincoln (10)

## C

Cameron, Dwight, Rockend Rd., Northeast Harbor (5)  
 Campbell, Fred G., Box 484, Warren (7)  
 Canal, Ory D., Augusta State Hospital, Augusta (6)  
 Capron, Charles W., 22 Bramhall St., Portland (3)  
 Carde, Albert M., 33 Elm St., Milo (11)  
 Carrier, John W., Central Maine Gen. Hosp., Lewiston (1)  
 Carson, Robert S., 11 McKeen St., Brunswick (3)  
 Carton, Arthur K., Market Square, Houlton (2)  
 Casey, William L., 131 State St., Portland (3)  
 Castellanos, Jose, Augusta State Hospital, Augusta (6)  
 Caswell, John A., 16 Waldo Ave., Belfast (13)  
 Chamberlin, Richard T., 14 Gilman St., Waterville (6)  
 Chapin, Milan A., 237 Turner St., Auburn (1)  
 Charest, Leandre R., 314 Alfred St., Biddeford (15)  
 Chase, George O., Eastern Maine Gen. Hosp., Bangor (10)  
 Chason, Sidney, 128 Broadway, Bangor (10)  
 Chasse, Richard L., 18 Park St., Waterville (6)  
 Chatterjee, Manu, 11 McKeen St., Brunswick (3)  
 Chen, Jen-Ti, Cherry Hill Terrace, Waterville (6)  
 Chenery, Frederick L., Jr., Monmouth (1)  
 Christensen, Harry E., South Freeport (3)  
 Ciampi, Louis A., Gray (3)  
 Clapp, Waldo A., 215 College St., Lewiston (1)  
 Clapperton, Gilbert, 300 Main St., Lewiston (1)  
 Clark, Frederick B., 131 State St., Portland (3)  
 Clark, Richard L., Freeport Medical Ctr., Freeport (3)  
 Clarkin, Charles P., 64 Brookside Rd., Portland (3)  
 Clement, James D., Jr., 77 Essex St., Bangor (10)  
 Clough, Dexter J., 2nd, 224 State St., Bangor (10)  
 Clough, Herbert T. (Col.), Hq. USAF (AFCSG 12),  
 Bldg. T-8, Washington 25, D. C. (10)  
 Cloutier, Wilfrid A., 646 Main St., Lewiston (1)  
 Cobb, Norman E., 132 Main St., Belfast (13)  
 Cobb, Stephen A., 34 Winter St., Sanford (15)  
 Coffin, Ernest L., Northeast Harbor (5)  
 Cole, Donald P., 45 Deering St., Portland (3)  
 Colley, Maynard B., 14 Main St., Farmington (4)  
 Collins, H. Douglas, Caribou Clinic, Caribou (2)  
 Cook, Aaron, 23 High St., Waterville (6)  
 Cooper, Llewellyn W., 194 Main St., Bar Harbor (5)  
 Cornell, Robert C., 118 Forest Ave., Orono (10)  
 Coulton, Donald, 326 State St., Bangor (10)  
 Covert, Stanley B., Kingfield (4)  
 Cox, William V., 133 Court St., Auburn (1)  
 Cragin, Charles L., 829 Congress St., Portland (3)  
 Craig, Allan, 905 Egan Ave., Pacific Grove, Calif. (10)  
 Crane, Lawrence, 157 Pine St., Portland (3)  
 Crawford, Albert S., East Blue Hill (6)  
 Crawford, J. Ramser, 105 Water St., Augusta (6)  
 Crawford, Joseph R., 105 Water St., Augusta (6)  
 Cross, Harold D., Main Rd. & Summer St.,  
 Hampden Highlands (10)  
 Crowe, James H., 57 Main St., Ellsworth (5)  
 Cruickshank, Frank S., Jr., 22 Forest St., Bar Harbor (5)  
 Cummings, George O., Sr., 47 Deering St., Portland (3)  
 Cummings, George O., Jr., 47 Deering St., Portland (3)  
 Cuneo, Kenneth J., 31 Summer St., Kennebunk (15)  
 Cunningham, Alice N., 32 Federal St., Brunswick (3)  
 Curran, Edward L., 209 State St., Bangor (10)  
 Curtis, John B., 10 High St., Milo (11)  
 Cutler, Lawrence M., 31 Grove St., Bangor (10)

## D

Dachslager, Philip, 21 Western Ave., Augusta (6)  
 Dalrymple, Sidney C., So. Great Rd., So. Lincoln, Mass. (8)  
 D'Andrea, Anthony L., 131 State St., Portland (3)  
 Daniels, Donald H., R.R. No. 1, Readfield (3)  
 Darlington, Brinton T., Doctors Park,  
 89 Hospital St., Augusta (6)  
 Davidson, David, 235 State St., Portland (3)  
 Davidson, Gisela K., 235 State St., Portland (3)  
 Davies, Lloyd G., 78 Main St., Fryeburg (3)  
 Davis, Ansel S., Springvale (15)  
 Davis, Earle M., 2 School St., Waterville (6)  
 DeCosta, Donald A., Poland Spring (1)

Defoe, Garfield G., Dixfield (9)  
 De la Garza, Alexander M., 111 Webster St., Lewiston (1)  
 Denison, John D., 105 Brunswick Ave., Gardiner (6)  
 Dennett, Carl G., 258 Main St., Saco (15)  
 Dennis, Richard H., 33 College Ave., Waterville (6)  
 Dennison, Frederick C., 183 Main St., Thomaston (7)  
 Derry, G. Hermann, 690 Congress St., Portland (3)  
 Desjardins, Richard F., 240 Penobscot Ave., Millinocket (10)  
 Devan, Thomas A., 10245-47th Ave., Corona, L. I., N. Y. (10)  
 DeWitt, James C., Address unknown (10)  
 Dienst, Stanley G., 190 Pine St., Portland (3)  
 Dietrich, Mary M., P.O. Box 93, Orrington (10)  
 Dionne, Maurice J., Baribeau Dr., Brunswick (3)  
 Dionne, William E., 75 Main St., Springvale (15)  
 Dixon, Walter G., 16 Deering St., Norway (9)  
 Doble, Miriam, 990 Washington St., Bath (8)  
 Doby, Tibor, 131 State St., Portland (3)  
 Donahue, Clement L., 22 Sweden St., Caribou (2)  
 Donahue, Gerald H., 4 Station St., Presque Isle (2)  
 Dooley, Francis M., 53 Deering St., Portland (3)  
 Dore, Clarence E., 2 School St., Waterville (6)  
 Dore, Kenneth E., 133 Main St., Fryeburg (3)  
 Dorogi, Louis V., 149 Main St., Freeport (3)  
 Dougherty, John F., 112 Front St., Bath (8)  
 Douphinett, Otis J., 763 Congress St., Portland (3)  
 Downing, J. Robert, 35 Summer St., Kennebunk (15)  
 Drake, Emerson H., 18 Bramhall St., Portland (3)  
 Draxler, James E., Ward Town Rd., Freeport (3)  
 Drummond, S. Dunton, Bar Mills (15)  
 Duffey, Richard V., 187 North Main St., Brewer (10)  
 Duffy, Wallace H., 100 Main St., Farmington (4)  
 Dunham, Carl E., 188 State St., Portland (3)  
 Dunham, Marguerite C., P.O. Box 748, Presque Isle (2)  
 Dunham, Rand A., P.O. Box 400, East Millinocket (10)  
 Dunn, Robert H., Veterans Administration, Togus (6)  
 Dwyer, Clement S., 205 French St., Bangor (10)  
 Dycio, George, 55 Broad St., Auburn (1)  
 Dycio, Mary T., 3 Bayberry Lane, Lewiston (1)  
 Dyhrberg, Norman E., 323 Main St., Cumberland Mills (3)

## E

Earle, Ralph P., Vinalhaven (7)  
 Earnhardt, Joseph B., 55 Strondwater St., Westbrook (3)  
 Eastman, Charles W., 15 Millet St., Livermore Falls (4)  
 Eddy, Robert H., 5 Beech St., Rockland (7)  
 Elmore, Dexter E., 11 Main St., Dixfield (9)  
 Emanuel, Meyer, Veterans Administration, Togus (6)  
 Emerson, W. Merritt, 131 State St., Bangor (10)  
 Emery, Frederick C., 242 Cedar St., Bangor (10)  
 Endicott, Ruth E., 16 Main St., Ogunquit (15)  
 English, Lena M., 489 Castle Shannon Blvd.,  
 Pittsburgh, Pa., 15234 (6)  
 Eppinger, Ernst, 52 Belmont St., Portland (3)  
 Ervin, Edmund N., 2 School St., Waterville (6)  
 Etscovitz, Eli A., Cary Memorial Hospital, Caribou (2)  
 Eyerer, Rudolf E., 489 State St., Bangor (10)

## F

Fagone, Francis A., 312 Congress St., Portland (3)  
 Faucher, Francois J., Grand Isle (2)  
 Feeley, J. Robert, 438 Garland St., Bangor (10)  
 Feiges, Lewis M., 331 Veranda St., Portland (3)  
 Ferguson, Andrew, 128 Broadway, Bangor (10)  
 Ferguson, Barbara, 80 Goff St., Auburn (1)  
 Ferguson, Franklin F., 22 Bramhall St., Portland (3)  
 Fichtner, Paul A., 781 High St., Bath (8)  
 Ficker, Robert F., Maine St., Kennebunkport (15)  
 Fife, James L., Baribeau Dr., Brunswick (3)  
 Finks, Henry B., 73 Deering St., Portland (3)  
 Fiorica, Gaetano T., 12 Church St., Chisholm (4)  
 Fish, Nicholas, 235 State St., Portland (3)  
 Fisher, Dean H., State House, Augusta (6)  
 Fisher, Samson, 173 Main St., Waterville (6)  
 Fishman, Louis N., 185 Webster St., Lewiston (1)  
 Flanders, Merton N., 370 Main St., Lewiston (1)



Floyd, Paul E., 2 Middle St., Farmington (4)  
 Fogg, C. Eugene, 35 Deering St., Portland (3)  
 Fogg, Philip S., Jr., 173 Pleasant Ave., Portland (3)  
 Fortier, Andre P., 68 Foss St., Biddeford (15)  
 Fortier, Paul J., 111 Webster St., Lewiston (1)  
 Foster, Thomas A., 131 State St., Portland (3)  
 Fox, Francis H., 83 West St., Portland (3)  
 Freeman, William E., 107 Main St., Yarmouth (3)  
 French, Rowland B., 16 Water St., Eastport (14)  
 Frenette, Francis F., 13479 Clifton Blvd.,  
 Lakewood 7, Ohio (2)  
 Friend, John W., 49 Hampton Ave., Auburn (1)  
 Frost, Harold M., Friendship (7)  
 Frost, Robert A., 93 Summer St., Auburn (1)  
 Fuller, Barbara L., 20 Chestnut St., Rockland (7)

## G

Gaillard, Richard A., 276 State St., Bangor (10)  
 Galen, Robert S., 6 Breckan St., Brunswick (3)  
 Garcia-Rey, Felix M., 14 Charles St., Milo (11)  
 Gates, Clifford W., 130 Main St., Gorham (3)  
 Gauvreau, Horace L., 82 Pine St., Lewiston (1)  
 Gauvreau, Norman O., 78 Pine St., Lewiston (1)  
 Geer, Charles R., 690 Congress St., Portland (3)  
 Geer, George L., Jr., 690 Congress St., Portland (3)  
 Getchell, Ralph A., 14 Elmwood Ave., Cape Elizabeth (3)  
 Geyerhahn, George, 73 Deering St., Portland (3)  
 Gibbons, John F., 22 Bramhall St., Portland (3)  
 Giberson, Raymond G., 156 A Academy St., Presque Isle (2)  
 Giddings, Lane, 6 E. Chestnut St., Augusta (6)  
 Giddings, Paul D., 31 Western Ave., Augusta (6)  
 Giesen, Joseph H., 34 Gilman St., Waterville (6)  
 Giguere, Eustache N., 90 Webster St., Lewiston (1)  
 Giguere, Leandre W., 30 Elm St., Waterville (6)  
 Gilman, Herbert C., 240 Penobscot Ave., Millinocket (10)  
 Gingras, Adolphe J., 105 Water St., Augusta (6)  
 Gingras, Napoleon J., 6 East Chestnut St., Augusta (6)  
 Glassmire, Charles R., 58 Deering St., Portland (3)  
 Goduti, Richard J., 9 Deering St., Portland (3)  
 Godsoe, John A., 19 Deering St., Portland (3)  
 Goldfarb, Jaime, Box C, Pownal (3)  
 Goldman, Morris E., 185 Webster St., Lewiston (1)  
 Goldman, Richard N., 185 Webster St., Lewiston (1)  
 Good, Philip G., 38 Deering St., Portland (3)  
 Goodof, Irving L., Thayer Hospital, Waterville (6)  
 Goodrich, Blynn O., 165 Main St., Waterville (6)  
 Goodwin, Ralph A., Sr., 56 Denison St., Auburn (1)  
 Goodwin, Ralph A., Jr., 33 Court St., Auburn (1)  
 Gormley, Eugene G., Market Square, Houlton (2)  
 Gould, George L., 79 Main St., Richmond (6)  
 Graves, Robert A., Sunset Drive, Orono (10)  
 Gray, Philip L., Blue Hill (5)  
 Greco, Edward A., 12 Pine St., Portland (3)  
 Green, Ross W., 33 Court St., Auburn (1)  
 Greene, John P., 19 Sabattus St., Lewiston (1)  
 Greene, Merrill S. F., 466 Main St., Lewiston (1)  
 Greenlaw, William A., 129 Main St., Fairfield (12)  
 Gregory, Frederick J., So. Main St., Caribou (2)  
 Gregory, Philip O., St. Andrews Hosp., Boothbay Harbor (8)  
 Griffin, Carl R., Jr., 69 Townsend Ave., Boothbay Harbor (8)  
 Griffiths, Eugene B., 429 Main St., Presque Isle (2)  
 Grimes, Gilbert R., 185 Webster St., Lewiston (1)  
 Grish, Albert J., Box C, Pownal (3)  
 Grow, William B., Central Maine Sanatorium, Fairfield (12)  
 Guillemette, Maurice R., 109 Water St., Augusta (6)  
 Guite, L. Armand, 45 Elm St., Waterville (6)

## H

Haas, Carl M., 357 Elm St., Biddeford (15)  
 Haas, Rudolph, 480 Main St., Lewiston (1)  
 Hall, Walter D., 407 Main St., Rockland (7)  
 Hall, Walter L. H., 130 Middle St., Old Town (10)  
 Hallett, George W., Jr., 72 West St., Portland (3)  
 Hamilton, Virginia C., So. Harpswell (8)  
 Hamlin, Irvin E., Main St., East Millinocket (10)

Hanley, Daniel F., P.O. Box 637, Brunswick (3)  
 Hannigan, Charles A., 85 Goff St., Auburn (1)  
 Hannigan, Margaret H., 85 Goff St., Auburn (1)  
 Harkins, Michael J., 437 Main St., Lewiston (1)  
 Harlow, Edwin W., 177 Main St., Waterville (6)  
 Harper, Harry L., 17 Main St., South Paris (9)  
 Harrison, George J., Market Sq., Houlton (2)  
 Harvey, Thomas G., 59 Mayo St., Caribou (2)  
 Harvey, William C., 59 Mayo St., Caribou (2)  
 Hawkes, Richard S., 47 Deering St., Portland (3)  
 Hawkins, Donald B., Atlantic Ave. and Sea St., Camden (7)  
 Hayward, I. Mead, So. Main St., Caribou (2)  
 Hazzard, Lawrence R., Breakfast Hill Rd.,  
 Greenland, N. H. (15)  
 Heath, Parker, Jr., 22 White St., Rockland (7)  
 Hecht, Henry, 326 Stevens Ave., Portland (3)  
 Hedin, Carl J., Penobscot Terrace, Brewer (10)  
 Heifetz, Ralph, 173 State St., Portland (3)  
 Helfrich, Harry M., Jr., 122 Academy St., Presque Isle (2)  
 Helfrich, Nancy R., 48 Third St., Presque Isle (2)  
 Herrick, Stanley E., Jr., 12 Deering St., Portland (3)  
 Herring, Leon D., Memorial Dr., Winthrop (6)  
 Herson, Joseph H., 343 E. 30th St.,  
 New York, N. Y., 10016 (2)  
 Hiebert, Clement A., 18 Bramhall St., Portland (3)  
 Hiebert, Joelle C., Jr., Box 148, Norway (9)  
 Higgins, George F., 122 Academy St., Presque Isle (2)  
 Higgins, George L., 15 Water St., Newport (10)  
 Hill, Allison K., 113 Somerset St., Bangor (10)  
 Hill, Douglas R., 855 Sawyer St., South Portland (3)  
 Hill, Frederick T., Thayer Hospital, Waterville (6)  
 Hill, Howard F., 33 College Ave., Waterville (6)  
 Hill, Kevin, 33 College Ave., Waterville (6)  
 Hill, Paul S., Jr., 323 Main St., Saco (15)  
 Hinckley, Harris, 331 Cottage Rd., South Portland (3)  
 Hirschberger, Celia, 44 Main St., Waterville (6)  
 Hoch, Gretl J., Box 146, Jackman (12)  
 Hochschild, Hugo, 33 Main St., Thomaston (7)  
 Hoffman, Alvin A., P.O. Box 222, York (15)  
 Hogan, Chester F., 62 Main St., Houlton (2)  
 Holt, C. Lawrence, 27 Deering St., Portland (3)  
 Holz, Peter H., 51 Elm St., Camden (7)  
 Hopkins, Herbert J., 24 Portland Ave., Old Orchard (15)  
 Hopping, John S., R.F.D. No. 2, Union (7)  
 Hornberger, H. Richard, 2 School St., Waterville (6)  
 Hornstein, Louis S., 220 Water St., Skowhegan (12)  
 Horsman, Donald H., 50 Goff St., Auburn (1)  
 Houle, Marcel P., 200 Alfred St., Biddeford (15)  
 Houlihan, John S., 209 State St., Bangor (10)  
 Howard, Emery B., 23A Summer St., Rockland (7)  
 Howard, George C., Oak St., Guilford (11)  
 Howard, Henry M., 105 Franklin St., Rumford (9)  
 Hsu, Theodore S., 14 High St., Ellsworth (5)  
 Hubbard, Roswell E., Waterford (9)  
 Hudson, Henry A., R.F.D. #1, West Bridgton (3)  
 Hughes, Edward J., Jr., 336 Mt. Hope Ave., Bangor (10)  
 Humphreys, Ernest D., 91 Main St., Pittsfield (12)  
 Hunter, Albert L., Knox County Gen. Hosp., Rockland (7)  
 Huntress, Roderick L., 988 Sawyer St., South Portland (3)  
 Hurd, Allan C., 72 Church St., Gardiner (6)  
 Hurwitz, Alfred, 32 Deering St., Portland (3)  
 Hutchins, Dean L., Health Department,  
 Univ. of Maine, Orono (10)

## I

Irwin, Carl W., 262 State St., Bangor (10)  
 Ives, Howard R., 131 Chadwick St., Portland (3)

## J

Jackson, Norman M., 151 Franklin St., Rumford (9)  
 Jacob, Donald R., Princeton (14)  
 Jacobson, Payson B., 295 Brighton Ave., Portland (3)  
 James, Chakmakis, 47 Howe St., Lewiston (1)  
 James, John A., 117 Goff St., Auburn (1)  
 Jameson, C. Harold, Medical Arts Building, Rockland (7)  
 Jan, M. Rafiq, 1065 Forest Dr., Ancora, N. J. (6)

Jellerson, Leon R., 77 Franklin St., Boston 12, Mass. (15)  
 Johnson, Albert C., 131 Chadwick St., Portland (3)  
 Johnson, Gordon N., P.O. Box 86, Houlton (2)  
 Johnson, James H., Jr., Pine St., Centerville, Mass. (11)  
 Johnson, Oscar R., 18 Deering St., Portland (3)  
 Johnson, R. Paul, Main St., Fort Kent (2)  
 Johnston, James S., York Harbor (15)  
 Jones, Paul A., Sr., Union (7)  
 Jones, Paul A., Jr., 2 School St., Waterville (6)  
 Joost, Arthur M., Jr., P.O. Box B, Bucksport (5)  
 Jordan, W. Edward, Jr., 68 Water St., Skowhegan (12)

## K

Kadi, Francis J., Bangor State Hospital, Bangor (10)  
 Kagan, Samuel H., 283 Water St., Augusta (6)  
 Kangas, Onni C., 417 Main St., Rockland (7)  
 Kay, Edwin, 31 Frye St., Lewiston (9)  
 Kazutow, John, Box 113, Columbia Falls (14)  
 Kellogg, Robert O., 316 State St., Bangor (10)  
 Kemezys, Kestutis M., 25 Garfield St., Madison (12)  
 Kent, Stanley W., 42 Deering St., Portland (3)  
 Kershner, Warren E., 57 Green St., Bath (8)  
 Kibbe, Frank W., R.F.D., Lincolnville (7)  
 Kimball, Herrick C., P.O. Box 372, Fort Fairfield (2)  
 Kinder, Edward L., Jr., 1027 Washington St., Bath (8)  
 King, Merrill J., Sr., 9 Deering St., Portland (7)  
 King, Merrill J., Jr., 22 White St., Rockland (7)  
 Kinghorn, Charles W., 4 Wentworth St., Kittery (15)  
 Kirk, William V., Eagle Lake (2)  
 Knickerbocker, Charles H., 15 High St., Bar Harbor (5)  
 Knowles, Robert M., 49 Deering St., Portland (3)  
 Konecki, John T., St. Mary's Hospital, Lewiston (1)  
 Kopfmann, Harry, Deer Isle (5)  
 Kudisch, Leonidas B., 11 Franklin St., Rumford (9)  
 Kunkle, E. Charles, 27 Deering St., Portland (3)  
 Kurzbard, Stephen J., AFES, 500 Forest Ave., Portland (3)

## L

Labbe, Onil B., Van Buren (2)  
 LaCasce, Joseph H., 50 Union St., Ellsworth (5)  
 LaFlamme, Paul J., 106 Russell St., Lewiston (1)  
 LaFond, Robert S., 258 Main St., Saco (15)  
 Lagace, Alfred F., Cary Mem. Hosp., Caribou (2)  
 Lambdin, Morris A., Maine Coast Mem. Hosp., Ellsworth (5)  
 Landwehr, George R., 21 Western Ave., Augusta (6)  
 Lane, Russell M., Water St., Blue Hill (5)  
 Laney, Richard P., 50 Water St., Skowhegan (12)  
 Langer, Ella, State House, Augusta (6)  
 Lansing, Peter F., 16 Macomber Ave., Augusta (6)  
 Lape, C. Philip, 131 Chadwick St., Portland (3)  
 Lapirow, Harry, 99 Main St., Kennebunk (15)  
 Lappin, John J., 171 State St., Portland (3)  
 Larochele, Joseph R., 42 Bacon St., Biddeford (15)  
 Larrabee, Charles F., 48 Mt. Desert St., Bar Harbor (5)  
 Larson, Karl V., East Machias (14)  
 Laughlin, K. Alexander, 201 State St., Portland (3)  
 Lawry, Oram R., Jr., 96 Limerock St., Rockland (7)  
 Leary, Gerald C., 144 State St., Portland (3)  
 Leddy, Percy A., Main St., Seal Harbor (10)  
 Lee, Kong, 22 Glenn Dr., Woodbury, L. I., N. Y. (10)  
 Leigh, Kenneth E., Brixham Rd., York (15)  
 Leighton, Wilbur F., 192 State St., Portland (3)  
 Leiter, Laban W., 175 Vaughan St., Portland (3)  
 Leitman, Reuben, 188 Sabattus St., Lewiston (1)  
 Lenfest, Stanley R., Waldoboro (8)  
 Lepore, Anthony E., 72 Church St., Gardiner (6)  
 Lesieur, Louis C., 255 Beach St., Saco (15)  
 Levy, Richard A., Maine Medical Ctr., Portland (3)  
 Libby, Harold E., 702 Main St., Westbrook (3)  
 Lichter, Horacio A., 54 Pine St., Lewiston (1)  
 Lidstone, Frederick B., 117 Goff St., Auburn (1)  
 Lieberman, Arthur N., 180 Broadway, Bangor (10)  
 Lightbody, Charles H., No. Main St., Guilford (11)  
 Lincoln, John R., 22 Bramhall St., Portland (3)  
 Lincourt, Armand S., 122 Main St., Sanford (15)

Loewenstein, George, Chebeague Island (7)

Winter Address — Aripeka, Florida

Logan, G. E. C., 131 State St., Portland (3)  
 Lombard, Reginald T., 793 Main St., South Portland (3)  
 Lopez, Eduardo A., 33 College Ave., Waterville (6)  
 Lord, Edwin M., 39 Franklin St., Rumford (9)  
 Lord, George A., 34 Winter St., Sanford (15)  
 Lord, Maurice E., Box 537, Lake Placid, Florida (12)  
 Lorentz, John J., Hyde Rehabilitation Ctr., Bath (15)  
 Lorimer, Robert V., 169 State St., Portland (3)  
 Love, Robert B., 97 Main St., Gorham (3)  
 Lovely, David K., 46 Deering St., Portland (3)  
 Lynn, Geraldine, 188 Russell St., Lewiston (1)

## M

MacBride, Robert G., 25 Washington St., Lubec (14)  
 Macdonald, Donald F., 263 State St., Bangor (10)  
 MacDonald, Lewis V. A., Main St., Washburn (2)  
 MacDougal, Wilbur E., 186 Nowell Rd., Bangor (11)  
 MacDougall, James A., 303 Penobscot St., Rumford (9)  
 Mack, Francis X., 144 State St., Portland (3)  
 MacVane, William L., Jr., 211 State St., Portland (3)  
 Madigan, John B., Houlton (2)  
 Magaouda, Michael M. P., 39 Old Orchard St.,  
 Old Orchard Beach (15)  
 Magocsi, Alexander W., York (15)  
 Mahaney, William F., 338 Main St., Saco (15)  
 Maier, Paul, 723 Congress St., Portland (3)  
 Maltby, George L., 31 Bramhall St., Portland (3)  
 Mann, David V., 22 White St., Rockland (7)  
 Manol, Jack, 157 Pine St., Portland (3)  
 Manter, Wilbur B., 1 Fern St., Bangor (10)  
 Marquardt, Matthias, Augusta State Hospital, Augusta (6)  
 Marshall, Donald F., 142 High St., Portland (3)  
 Marshall, Joseph A., 177 Main St., Waterville (6)  
 Marshall, Richard A., 22 Bramhall St., Portland (3)  
 Marsters, David W., Phillips (4)  
 Marston, Henry E., North Anson (12)  
 Marston, Paul C., Kezar Falls (3)  
 Martel, Cyprien L., Jr., 91 Bartlett St., Lewiston (1)  
 Martin, Joseph E., 35 Main St., Mexico (9)  
 Martin, Ralf, 131 Chadwick St., Portland (3)  
 Martin, Thomas A., 157 Pine St., Portland (3)  
 Mason, Peter H., Millinocket Com. Hosp., Millinocket (10)  
 Mathews, Hugh J., Jr., 345 Water St., Gardiner (6)  
 Matthews, Edward C., 131 Chadwick St., Portland (3)  
 Mazzone, Giovanni, 487 Stevens Ave., Portland (3)  
 Melendy, Oakley A., Doctors Park, 89 Hospital St., Augusta (6)  
 Melkis, Andrew, Box C, Pownal (3)  
 Melnick, Jacob, 333 Congress St., Portland (3)  
 Memmelaar, Joseph E., 54 Forest Ave., Bangor (10)  
 Mendes, Joseph M., 5 School St., Lisbon Falls (1)  
 Mendros, John G., 111 Webster St., Lewiston (1)  
 Merriam, Thornton W., Jr., 44 James St., Bangor (10)  
 Merrill, Urban H., Etna (10)  
 Metcalf, John T., 47 Elm St., Milo (11)  
 Michaud, Joseph C., 6377 Eldredge Rd.,  
 Bedford Heights, Ohio (6)  
 Milazzo, John, 42 Elm St., Auburn (1)  
 Millard, Kathleen M. A., Windham Ctr. Rd., Windham (3)  
 Miller, Clark F., 46 Madison St., Auburn (1)  
 Miller, Hudson R., 11 Turner St., Auburn (1)  
 Miller, Thor, 752 Main St., Westbrook (3)  
 Milliken, Howard H., 105 Second St., Hallowell (6)  
 Millington, Paul A., 44 Mountain St., Camden (7)  
 Mills, Nathaniel, North Main St., Wolfeboro, N. H. (9)  
 Miragliuolo, Leonard G., 10 Maple St., Bangor (10)  
 Mitchell, Hazen C., Calais (14)  
 Mohlar, Robert G., 11 McKean St., Brunswick (3)  
 Monaghan, Stephen E., 157 Pine St., Portland (3)  
 Monkhouse, William A., 131 State St., Portland (3)  
 Monsivais, Alfredo, Augusta State Hospital, Augusta (6)  
 Moore, Beryl M., Oxford (9)  
 Moore, Valentine J., Thayer Hospital, Waterville (6)  
 Morin, Gerard L., 104 Ash St., Lewiston (1)  
 Morin, Harry F., 905 Middle St., Bath (8)  
 Morissette, Russell A., 185 Webster St., Lewiston (1)



Morris, Craig W., 50 Bangor St., Augusta (6)  
 Morrison, Alvin A., 57 Deering St., Portland (3)  
 Morrison, Robert M., 148 State St., Portland (3)  
 Morse, Edward K., 22 White St., Rockland (7)  
 Moulton, Albert W., Sr., 180 State St., Portland (3)  
 Moulton, Albert W., Jr., 180 State St., Portland (3)  
 Moulton, Gardner N., 5 Grove St., Bangor (10)  
 Moulton, Marion K., West Newfield (15)  
 Munce, Richard T., 262 State St., Bangor (10)  
 Mundie, Perley J., 32 North St., Calais (14)  
 Murphy, John J., 84 Portland St., South Berwick (15)  
 Myer, John C., Nasson College, Springvale (15)

## Mc

McAdams, William R., 723 Congress St., Portland (3)  
 McAllister, John W., 39 Water St., Lubec (14)  
 McCann, Eugene C., 49 Deering St., Portland (3)  
 McCormack, Roland L., 12 Bridge St., Norway (9)  
 McCrum, Philip H., 188 State St., Portland (3)  
 McEvoy, Charles D., Jr., 186 State St., Bangor (10)  
 McFarland, Edward A., Baribeau Dr., Brunswick (3)  
 McGinn, John F., 205 French St., Bangor (10)  
 McIntire, Barron F., Jr., 13 W. Elm St., Yarmouth (3)  
 McIntire, Percy, Central Maine Sanatorium, Fairfield (12)  
 McIntyre, John D., 50 Union St., Ellsworth (5)  
 McKay, Roland L., P.O. Box 265, Augusta (6)  
 McLaren, John J., Baribeau Dr., Brunswick (8)  
 McLarn, William D., 50 Union St., Ellsworth (5)  
 McLaughlin, Clarence R., 345 Water St., Gardiner (6)  
 McLaughlin, Ivan E., 345 Water St., Gardiner (6)  
 McLean, E. Allan, 29 Deering St., Portland (3)  
 McLean, Preston A., 336 Mt. Hope Ave., Bangor (10)  
 McLellan, William A., 87 Chestnut St., Camden (7)  
 McManamy, Eugene P., 72 West St., Portland (3)  
 McMichael, Morton, 73 Deering St., Portland (3)  
 McNamara, Wesley C., 8 Lee St., Lincoln (10)  
 McNeil, Harry D., 81 Silver Rd., Bangor (10)  
 McQuillan, Arthur H., 177 Main St., Waterville (6)  
 McQuoid, Robert M., 39 Columbia St., Bangor (10)

## N

Nackley, George N., 1 School St., Machias (14)  
 Nadeau, J. Paul, 91 Pine St., Lewiston (1)  
 Nadeau, Lawrence A., 41 Sherbrooke Ave., Lewiston (1)  
 Nangle, Thomas P., West Paris (9)  
 Nelson, Chesley W., 121 Main St., Norway (9)  
 Nelson, Isaac, Box 336, Greenville (11)  
 Nesin, Bourcard, 10 Water St., Howland (10)  
 Netland, Anders T., 317 State St., Bangor (10)  
 Newcomb, Charles H., Clinton (6)  
 Nicholas, Eric F., 11 Green St., Fort Fairfield (2)  
 Nichols, Arthur A., Edgecomb (8)  
 Nickerson, Norman H., Greenville (11)  
 Nielsen, Odd S., 85 Pleasant St., Dexter (11)

## O

O'Brien, William A., Arthur R. Gould Mem. Hosp.,  
 Presque Isle (2)  
 Oceretko, Arkadij, 37 Court St., Bath (8)  
 O'Connell, George B., 11 Lisbon St., Lewiston (1)  
 O'Connor, Francis J., 4 Woodlawn St., Augusta (6)  
 O'Donnell, Eugene E., 32 Deering St., Portland (3)  
 Oestrich, Alfred, 89 Congress St., Rumford (9)  
 Ohler, Robert L., Veterans Administration, Togus (6)  
 O'Kane, Francis R., 122 Penobscot Ave., Millinocket (10)  
 Olmsted, Burton L., 73 Deering St., Portland (3)  
 O'Meara, Edward S., Maine Coast Mem. Hosp., Ellsworth (5)  
 Onat, Mustafa V., St. George (7)  
 Orbeton, Everett A., 131 Chadwick St., Portland (3)  
 Osborne, John R., Narrows Pond Rd., Winthrop (2)  
 Osher, Harold L., 131 Chadwick St., Portland (3)  
 Osler, Jay K., 74 Birch St., Bangor (10)  
 O'Sullivan, William B., 331 Main St., Saco (15)

Ottum, Alvin E., 148 State St., Portland (3)  
 Ouellette, Benoit, 77 Main St., Fort Kent (2)  
 Ouellette, Marcel D., 114 Main St., Sanford (15)

## P

Page, Rosario A., 22 Sweden St., Caribou (2)  
 Palmer, Thomas H., Jr., 316 State St., Bangor (10)  
 Papadopoulos, George, Box 724, State Hospital, Augusta (6)  
 Parrot, Hadley, 74 Somerset St., Bangor (10)  
 Patane, Joseph M., 256 Alfred St., Biddeford (15)  
 Patterson, James, 1 Bay Rd., South Portland (3)  
 Patterson, John C., Box 724, State Hospital, Augusta (6)  
 Pawle, Robert H., 8 Walcott Ave., Falmouth (3)  
 Pearson, Henry, Brownfield (9)  
 Pearson, John J., 100 So. Main St., Old Town (10)  
 Peddie, Harry M. K., Doctors Park,  
 89 Hospital St., Augusta (6)  
 Pendleton, Arthur D., 3 Green St., Fort Fairfield (2)  
 Pennoyer, Douglass C., 112 Vaughan St., Portland (3)  
 Penta, Walter E., 316 Woodford St., Portland (3)  
 Perkins, Niles L., Jr., 16 Bramhall St., Portland (3)  
 Perrault, Oscar W., 30 South St., Biddeford (15)  
 Peterlein, Walter R., Jr., 75 Main St., Springvale (15)  
 Petterson, Herman C., Chebeague Island (3)  
 Pfeiffer, Paul H., 14 Gilman St., Waterville (6)  
 Philbrick, Maurice S., 292 Water St., Skowhegan (12)  
 Pines, Philip, Maine St., Limestone (2)  
 Platt, Anna, Beauchamp Rd., Rockport (7)  
 Winter Address — 110 Manatee Rd., Belleair,  
 Clearwater, Florida  
 Plimpton, Jay R., 283 Water St., Augusta (6)  
 Pogue, Jackson S., 529 Gilmore Ave., Trafford, Pa. (3)  
 Poliner, Irving J., 235 State St., Portland (3)  
 Polisner, Saul R., 143 Vaughan St., Portland (3)  
 Pomerleau, Ovid F., 179 Main St., Waterville (6)  
 Pomerleau, Rodolphe J. F., 27 Main St., Waterville (6)  
 Pooler, Harold A., State Hospital, Bangor (10)  
 Pope, W. Dean, 6 Pleasant St., Rangeley (4)  
 Porter, Edward C., 489 State St., Bangor (10)  
 Porter, Joseph E., 22 Bramhall St., Portland (3)  
 Potts, Ronald S., Central Maine Gen. Hosp., Lewiston (1)  
 Poulin, Albert A., Cherry Hill Dr., Waterville (6)  
 Poulin, James E., 177 Main St., Waterville (6)  
 Powell, Ralph C., Damariscotta (8)  
 Pratt, George L., 7 Main St., Farmington (4)  
 Pratt, Harold S., Livermore Falls (1)  
 Pratt, Loring W., 177 Main St., Waterville (6)  
 Price, Richard D., R.F.D. 2, Caribou (2)  
 Pritham, Fred J., Greenville Junction (11)  
 Proctor, Ray A., Garden Circle, Caribou (2)  
 Proctor, Thomas E., Boothbay Harbor (8)  
 Proudian, Paul O., 776 Main St., Westbrook (3)  
 Proulx, Harvey J., 92 Pine St., Lewiston (1)  
 Provost, Helen C., 48 Green St., Augusta (6)  
 Provost, Pierre E., 48 Green St., Augusta (6)  
 Purinton, William A., 276 State St., Bangor (10)

## R

Rand, Carleton H., 219 Oak St., Lewiston (1)  
 Rando, Joseph J., 111 Webster St., Lewiston (1)  
 Ray, Ferris S., 131 Chadwick St., Portland (3)  
 Read, Seth H., 15 Church St., Belfast (13)  
 Reed, Howard L., 68 Water St., Skowhegan (12)  
 Reed, James W., 18 Main St., Farmington (4)  
 Reel, John J., 59 So. Front St., Richmond (6)  
 Reeves, Edward L., 179 Sabattus St., Lewiston (1)  
 Reeves, Helene M., 179 Sabattus St., Lewiston (1)  
 Renzulli, Ottone, 346 Elm St., Biddeford (15)  
 Reynolds, Arthur P., 29 Second St., Presque Isle (2)  
 Reynolds, John F., 216 Main St., Waterville (6)  
 Reynolds, Ralph L., 216 Main St., Waterville (6)  
 Rice, William C., Main St., Calais (14)  
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 Richards, Carl E., 34 Winter St., Sanford (15)  
 Richards, Lee W., Jr., 21 Western Ave., Augusta (6)

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 Ridlon, Joseph R., 58 South St., Gorham (3)  
 Ridlon, Magnus F., 99 Broadway, Bangor (10)  
 Robert, Roger J. P., 331 Main St., Saco (15)  
 Robertson, George J., 171 Harrison Ave., Boston, Mass. (6)  
 Robinson, Hugh P., 27 Deering St., Portland (3)  
 Rock, Daniel A., 477 Main St., Lewiston (1)  
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 Rodriguez, Jose M., 109 Silver St., Waterville (6)  
 Root, John A., 22 White St., Rockland (7)  
 Ross, H. Danforth, 34 Winter St., Sanford (15)  
 Ross, Maurice, 372 Main St., Saco (15)  
 Roussin, William T., 48 Bacon St., Biddeford (15)  
 Rowe, Daniel M., Kirkwood Rd., Scarborough Beach (3)  
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 Russell, Theodore M., 21 Western Ave., Augusta (6)

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 Saunders, Norman W., 12 Deering St., Portland (3)  
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 Sawyer, Howard P., Jr., 22 Bramhall St., Portland (3)  
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 MD "B," Augusta (6)  
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 Shapiro, Morrill, 29 Deering St., Portland (3)  
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 Smith, Joseph I., 118 Front St., Bath (8)  
 Smith, Kenneth E., Veterans Administration, Togus (6)

Smith, Margaret S., Box 967, Presque Isle (2)  
 Smith, Oney P., Post Rd., Wells (15)  
 Somerville, Robert B., 45 Hillside St., Presque Isle (2)  
 Somerville, Wallace B., Mars Hill (2)  
 Sommerfeld, Kurt A., Veterans Adm., Togus (6)  
 Soroka, Selic, 39 High St., Skowhegan (12)  
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 Southworth, John D., Hartland (12)  
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 Viger, Leopold A., 176 Elm St., Biddeford (15)

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 Woodcock, John A., 35 Second St., Bangor (10)  
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 Young, John, Paradise St., Bethel (9)

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